The
Southern Surgeon

VOLUME IV AUGUST, 1935 NUMBER 4

SOME ATYPICAL CASES OF MALIGNANCY OF THE STOMACH

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THE mortality from cancer in the United States has increased by leaps and bounds since 1920. In 1920 there were 72,931 deaths from cancer; in 1933 there were 128,475. The chief captain of the men of death is cancer of the stomach. In 1920 there were 17,625 deaths from cancer of the stomach, and in 1933 the number of deaths from cancer of the stomach and duodenum, now grouped together in the United States Census reports, was 26,565.

In spite of this depressing statistical view, a little comfort may be derived from the fact that in the last three years the number of deaths from cancer of the stomach has not increased so far as the rate per 100,000 of population is concerned. In 1930 the number of deaths from this disease per 100,000 population was 21.4, and in 1933 it was 21.1. Although the number of deaths has actually increased, the population has also increased so that the ratio in the last three years has slightly diminished.

It has been estimated that the expectancy of life in the United States will be greater for the next twenty years, and even if there is no actual increase in the ratio of cancer deaths when arranged according to the decades of life, the fact that there will be more people in the later decades will probably result in more actual deaths from cancer. There are more persons being shoved into the far reaches of the "tropic of cancer."

The methods of curing cancer in its early stages are fairly

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Read before the Sixth Annual Assembly of The Southeastern Surgical Congress, in Jacksonville, March 11, 12 and 13, 1935.

satisfactory. Some cancers are amenable to irradiation with x-ray or radium. Others can be better treated by excision, and still others by a combination of irradiation and excision. The difficulty lies in making an early diagnosis. Cases of cancer, particularly cancer of the stomach, often reach the surgeon when the disease is too advanced to warrant radical operation. Cancer of the stomach is usually radio-resistant, and the only satisfactory treatment is excision.

A study of the stomach's physiology reveals large "silent" areas along its greater curvature and in the cardiac portion. A lesion in these areas, unless it perforates or bleeds, may give no symptoms whatever until it becomes sufficiently large to produce mechanical obstruction. This accounts for much of the delay. However, it seems probable that a more careful investigation of the apparently trivial symptoms of indigestion or dyspepsia, and an accurate detection of their causes, should lead to a correct diagnosis in some instances when failure to appreciate the importance of such slight digestive disturbances, especially in those of cancer age, would result in a fatal delay in the diagnosis. We need to follow the illustrious example of the late Sir James Mackenzie, and study disease in its incipiency, watch the little things that first appear in any patient who may be a subject of malignancy, and follow these symptoms through until their cause is definitely determined.

Balfour has said that he has about 50 per cent of five-year cures in those cancers of the stomach excised when they are still strictly confined to the stomach wall.

Gastric cancer is often described in the textbooks as a clinical entity, but it has no pathognomonic signs or symptoms. Gaither, in an excellent review of 245 cases, has shown that much of the textbook description of the symptoms of this disease is entirely useless. The lesion of the stomach which gives rather regular symptoms of peptic ulcer with periodic pain or discomfort, later becoming constant, is suggestive of cancer; but this change may arise from the perforation of a peptic ulcer or from adhesions. Pain of all kinds from slight discomfort to agonizing seizures may occur in the epigastrium in cancer of the stomach, and, on the other hand, cancer may develop in the cardiac portion to a size that almost fills the

lumen before any symptoms can be elicited. Fortunately, a cancer along the lesser curvature or about the pylorus where obstruction is produced gives earlier symptoms, and 42.2 per cent of cancers of the stomach occur at or near the pyloric end. Excessive bleeding is rare in cancer of the stomach, but vomiting a small amount of coffee-ground material may occur. The statistics of Warwick show that only about 50 per cent of cancers of the stomach ulcerate, which accounts for the infrequent bleeding. Occasionally excessive hemorrhage does occur, probably in less than 5 per cent of the cases, whereas there is marked hemorrhage in about 20 per cent of peptic ulcers.

When cancer of the stomach is suspected and cannot be excluded by a careful analysis of the symptoms, a thorough roentgenologic examination should be made. This is the best single method of diagnosing cancer of the stomach, and should be done by a competent roentgenologist who is accustomed to this work and is skilful in the use of the fluoroscope and in interpreting the plates.

I have selected for report a few unusual or atypical cases of gastic cancer that seem interesting:

CASE 1. A 70-year-old white woman complained of generalized abdominal pain with gas and nausea at infrequent intervals for fifteen years. A few months before admission the nausea became more frequent and the pain more constant and localized in the lower epigastrium. During two weeks before admission she vomited several times. The pain was dull and seemed to have little relation to meals; there was no hematemesis or melena. She was constipated and had lost about ten pounds in weight during a few months before admission. Roentgenologic examination showed a filling defect near the pylorus. On Dec. 10, 1928, partial gastrectomy was done.

The specimen measured 7 cm. along the lesser curvature and 8.3 cm. along the greater curvature. The diameter at the cardiac end was 7.5 cm., and at the duodenal end 4 cm. On section an oblong, slightly ulcerated lesion, 2 cm. in its longest diameter, was found on the lesser curvature toward the posterior wall about half-way between the pylorus and the cardiac end of the specimen. It infiltrated gradually into the tissues around it. Microscopic examination showed an area in which there were two distinctly carcinomatous acini in the midst of much inflammatory and repairing tissue. No other cancerous area was found.

The patient is well and without recurrence more than six years after the operation.

Can it be doubted that if we could get cases of cancer of the stomach as early as this we could cure a vast majority of them? This is a definite illustration of the change from a benign peptic ulcer into malignancy. No one can claim that this small

group of acini is not malignant for the structure of cancer here is obvious, and it would not be reasonable to suppose that they have been latent for fifteen years. Peptic ulcer of the duodenum is far more common than peptic ulcer of the stom-

FIG. 1. Mrs. R. H. B. Anterior view of specimen from partial gastrectomy and resection of the transverse colon. Note the tumor formation from the greater curvature, involving the mesentery of the transverse colon. (Case 3.)

ach and should be treated medically at first, particularly in the young, unless there are certain complications. While duodenal ulcer rarely becomes malignant, it is undoubtedly true that peptic ulcer of the stomach does become cancerous in some instances, and the only point for discussion is what per-

centage of peptic ulcers of the stomach undergo such change. Those who favor a low percentage claim that many of these ulcers supposed to undergo malignant change are cancerous from the beginning, and that this type of lesion really begins with a small ulcerating carcinoma.

But for the practical purpose of curing the cancer it can be easily seen that whether the lesion was from the first a low-

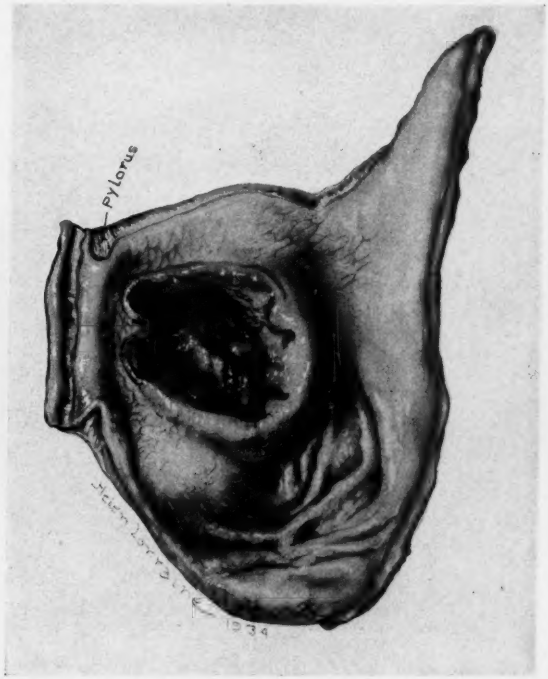

FIG. 2. Internal view of the tumor of the stomach in the preceding figure, the specimen being incised along the lesser curvature. The growth formed a pocket which had involved the mesocolon and the anterior abdominal wall. At operation the affected part of the abdominal wall was removed with the cautery. The lesion was on the greater curvature of the stomach, not obstructive, and consequently gave practically no symptoms until one month before she was admitted to the hospital. (Case 3.)

grade ulcerating cancer or a peptic ulcer that has become malignant, is merely an academic question. Any ulcer of the stomach that does not yield to medical treatment within a few weeks, as demonstrated clinically and by x-ray, should be considered cancerous. I use practically the same technic, partial gastrectomy, in ulcers of the stomach as I do in cancer.

The operative risk is apparently not increased, and the function of the stomach seems to be satisfactorily restored by the Billroth-I type of operation.

CASE 2. A 38-year-old white woman developed symptoms of gas and belching, but without nausea and vomiting, six months before admission. She had in the upper right abdomen a gnawing sensation with dull pain which was relieved by food or soda. She had been on a liquid diet for two weeks

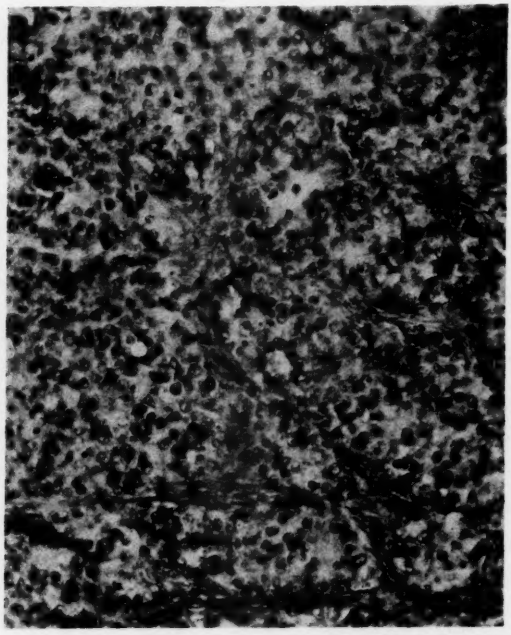

FIG. 3. Photomicrograph of tissues from the margin of the cancer shown in the preceding illustration. The growth is quite cellular and, while there is some necrosis, many of the cells are active and there are numerous mitotic figures. It may be classed as grade 3. (Case 3.)

before admission. On examination an indefinite mass was found in the mid-epigastrium. Roentgenograms showed a constant filling defect in the pyloric portion of the stomach. On May 15, 1929, a partial gastrectomy was done. In the specimen the lesion showed considerable infiltration in the submucosa and only a small superficial ulcer. On microscopic examination, however, there was an unusual type of malignancy that springs from the deeper layers of the gastric mucosa and does not tend to form acini. This was graded 3 by Broders. The patient recovered satisfactorily and lived four years without signs of recurrence. Finally, she developed a mass in the epigastrium, extensive metastases, and died March 25, 1934.

The interesting features about this case are that the patient was a comparatively young woman and the lesion showed a high grade of malignancy, a type that is believed to recur quite promptly. Yet she lived four years without symptoms of recurrence.

CASE 3. Mrs. R. H. B., aged 67, entered the hospital on Dec. 5, 1933, complaining of shortness of breath, loss of appetite and slight intestinal gas of one month's duration. There had been no pain, diarrhea, constipation or bloody stools. A mass in the right abdomen on a level with the umbilicus was found. Roentgenologic examination revealed a freely movable stomach and on the greater curvature near the pylorus a gross defect which was believed to be cancer.

At operation on Dec. 15, 1933, the mass, to which the stomach was adherent, was incised and it discharged foul, rather thick, light yellowish pus.

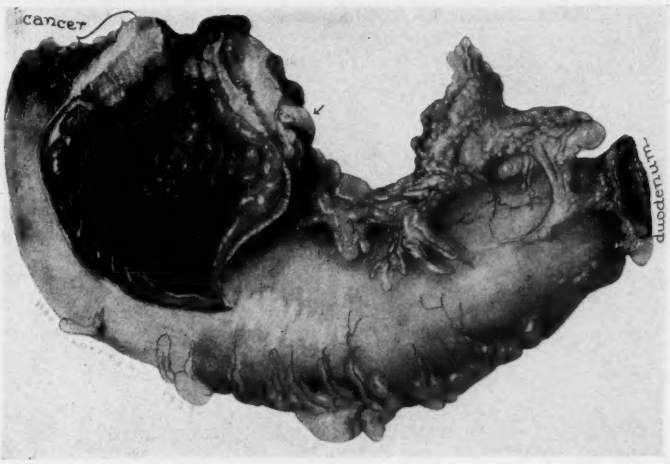

FIG. 4. Mrs. T. G. H. Specimen of the entire stomach, following a total gastrectomy. The stomach has been opened on the posterior wall. The lesion is along the cardiac portion and involves the lower end of the esophagus. (Case 4.)

There was apparently no bile or gastric content. She left the hospital about five weeks after operation feeling better and with a good appetite. Later she gradually became weaker, with frequent abdominal distention during the evening and eructations of gas, loss of appetite and nausea, and the passage of very dark colored stools. There was a mass, not tender on pressure, at the site of the previous abscess. An x-ray examination at this time showed the same filling defect in the stomach which was thought to indicate cancer.

A second operation on March 24, 1934, showed that the growth involved the mesentery of the transverse colon, so that resection of both the transverse colon and the stomach were necessary. (Figs. 1, 2 and 3.) We had given intra-abdominally Steinberg's vaccine the day before this operation, and I think it added something to her smooth convalescence. The patient at pres-

ent is well and without evidence of recurrence, although the histologic picture shows a rather malignant type of cancer.

CASE 4. Mrs. T. G. H., aged 48, had experienced gastric symptoms for only three and a half months before admission. In Philadelphia her esophagus

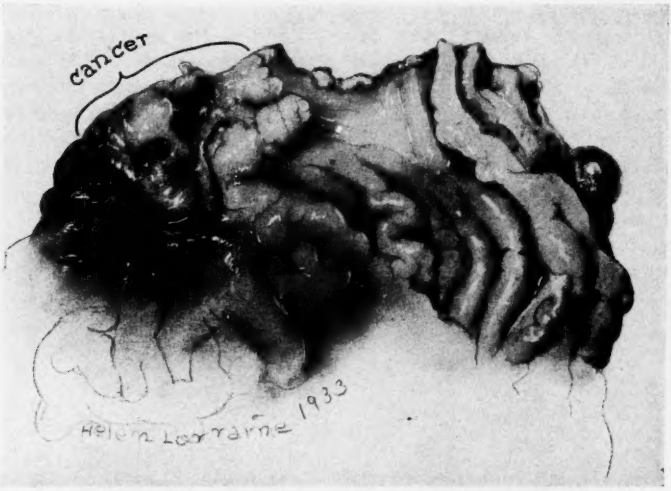

FIG. 5. Another view of the cancer shown in the preceding illustration, with the stomach opened more widely. The growth impinged upon and involved the lower portion of the esophagus. (Case 4.)

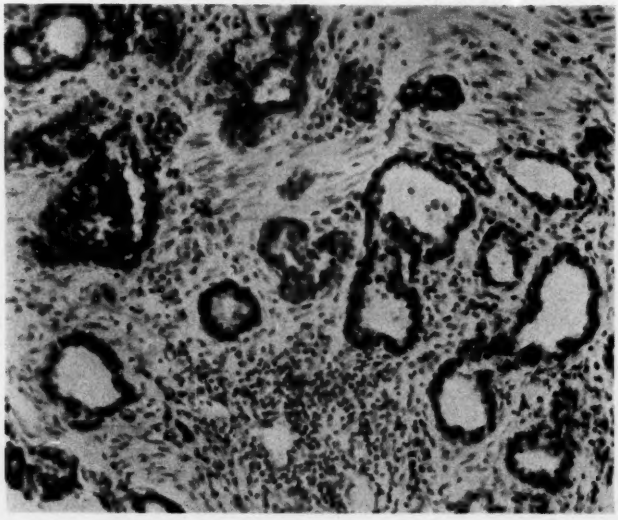

FIG. 6. Photomicrograph of the cancer shown in the two preceding drawings. The growth is adenocarcinoma. (Case 4.)

had been examined and a biopsy done, which showed cancer in the cardiac portion of the stomach. A total gastrectomy was performed. (Figs. 4, 5, 6 and 7). She made a satisfactory immediate recovery, but later developed pulmonary symptoms on the left side, and died twelve days after the operation. Necropsy showed a large, left subdiaphragmatic abscess. The lumens of the

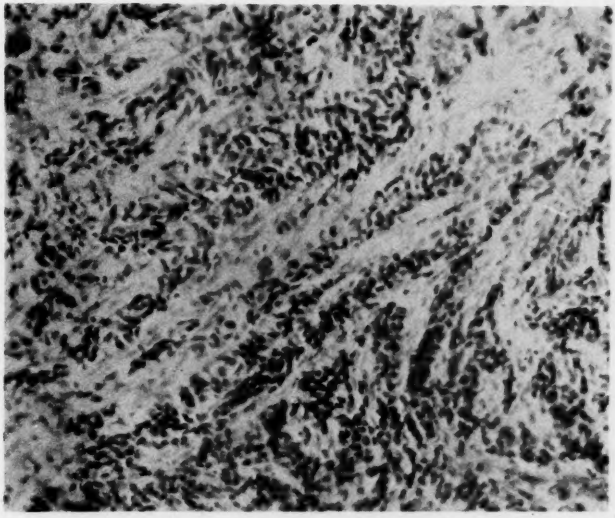

FIG. 7. Photomicrograph of another portion of the growth shown in Figures 4 and 5. The cells here are not well differentiated into acini and seem to indicate a higher grade of malignancy at this point than in the preceding photomicrograph. (Case 4.)

esophagus and jejunum were well open. Apparently the site of the anastomosis formed a wall of the abscess. There was also evidence of peritonitis in the lower part of the peritoneal cavity. The lower half of the left lung was congested and consolidated.

Following total gastrectomy it would be well to drain directly or with a long rubber tube extending from the region of the hilum of the spleen through a stab wound in the left flank, because serum may accumulate in the large cavity left in this area after operation, and stagnation with infection occur.

CASE 5. Mr. C. F. W., aged 31, had always been unable to eat certain foods because they produced heartburn. For five or six months before admission he had a tired feeling with discomfort in his stomach. These symptoms had not been severe enough to incapacitate him until five weeks before admission, when he began to have a gnawing pain which came on about three hours after each meal. This was relieved by food or milk; he had never taken soda. Two weeks before admission he had to stop work, and shortly after this he vomited at intervals for two days; on one occasion the vomitus con-

tained chocolate-colored material. Vomiting relieved the pain. He had lost 18 pounds in the previous five months. Dr. A. B. Moore, Washington, D. C., had examined him roentegenologically and reported an inoperable cancer of the stomach. From his age, however, and the fact that he had a high percentage of free hydrochloric acid in the gastric juice (fasting, 74°; 20 minutes, 73°; 40 minutes, 54°; 60 minues, 64°), I thought the lesion was probably a penetrating peptic ulcer. A partial gastrectomy was done on Sept. 26, 1933. A small ulcer was found near the pylorus and a large penetrating one in the posterior gastric wall with the base attached to the pancreas. A cautery dissection of the adherent portion of the pancreas was done so that the ulcer was not opened during the operation. There was no evidence of any other extra-gastric lesion.

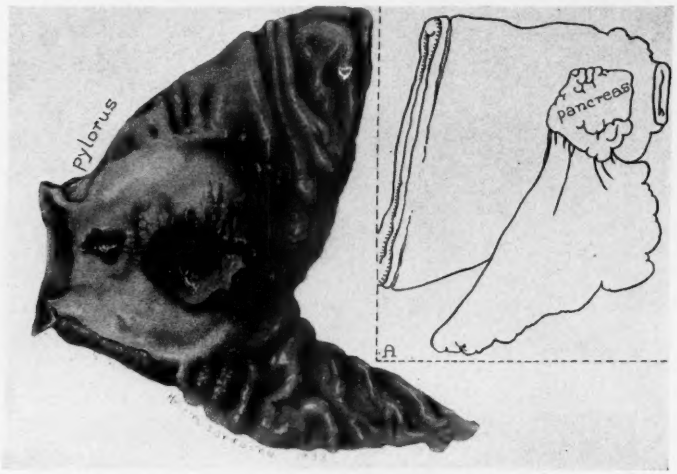

FIG. 8. Mr. C. F. W. Specimen removed by partial gastrectomy. A large excavating ulcer is seen on the posterior wall of the stomach. The base is necrotic, and consists of a portion of the pancreas which has been removed with the cautery without opening the ulcer. The margins of the ulcer are sharp and not everted except at one area. A smaller ulcer which has not entirely perforated is seen between the pylorus and the larger ulcer. Insert shows a posterior view with the attached pancreas which formed the base of the ulcer. The remaining portion of the posterior wall was apparently normal. (Case 5.)

Examination of the margin of the penetrating ulcer seemed to show only inflammatory tissue. (Figs. 8, 9, 10 and 11.)

The patient made a satisfactory immediate recovery, and felt much relieved. Seven months later he returned with a mass in the abdomen. A biopsy was done and malignancy found. The original specimen was again examined. Blocks taken from the base of the ulcer showed grade 4 small round cell carcinoma. X-ray treatment by Dr. Fred Hodges caused marked recession of the growth, until it almost disappeared, but rapidly it recurred and he died on June 21, 1934.

Necropsy showed extensive and abundant metastases of small round cell carcinoma throughout the abdomen, but the mucosa of the stomach and duodenum was smooth and unaffected.

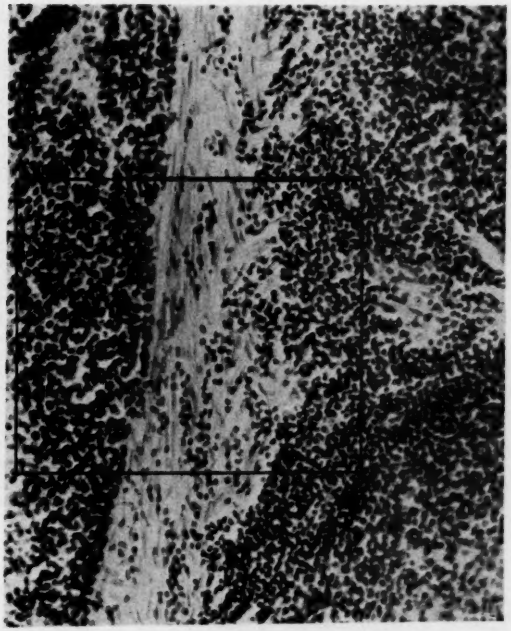

FIG. 9. Photomicrograph of tissue from the deeper portion of the ulcer shown in Fig. 8. On one side can be seen lymphocytes and the small round cells of inflammation. On the opposite side, separated by stroma of connective tissue, are the somewhat larger malignant cells constituting a small round cell carcinoma. Although the difference in the cells is obvious when they are observed here, the gross appearance of the lesion seems to indicate an inflammatory condition. (X 250.) (Case 5.)

Such cases emphasize the necessity of radical procedure in the treatment of gastric peptic ulcers, and careful histologic examination as they might easily be considered inflammatory.

CASE 6. Finally, I wish to report a case of lymphosarcoma of the stomach. Unlike most lesions of the stomach, lymphosarcoma is radio-sensitive. It is difficult to distinguish between carcinoma and lymphosarcoma of the stomach before operation, but as soon as possible after resection deep x-ray therapy should be employed. In this patient, who was operated upon nearly five years ago, the importance of giving this therapy was not appreciated. A 73-year-old white woman, seven months before admission, began to suffer from gas, tenderness and a dull aching pain in the epigastrium. These symptoms gradually became worse. At first there was no relation to meals, but later the discomfort was increased by food. She had vomited twice, both times following a dietary indiscretion. She had lost 15 or 20 pounds in weight during this time. There was an indefinite mass palpable in the upper portion of the abdomen. Roentgenologic examination showed a gross defect in the mid-portion of the stomach which was, however, movable. A partial gastrectomy was done on May 6, 1930. She made a satisfactory recovery.

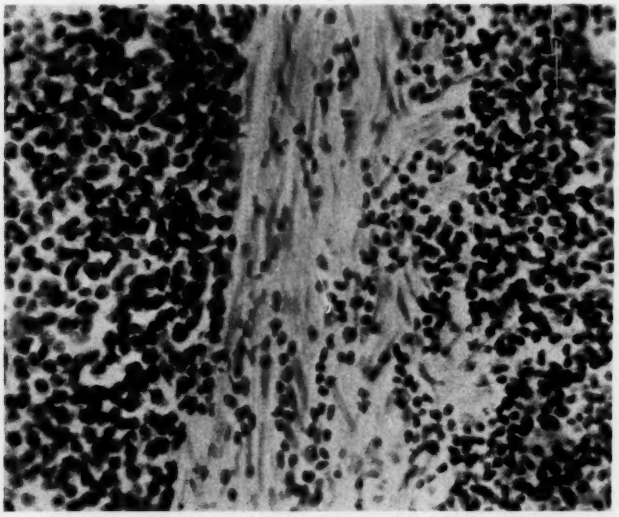

FIG. 10. A higher magnification of the field shown in Fig. 9. (X 450.) (Case 5.)

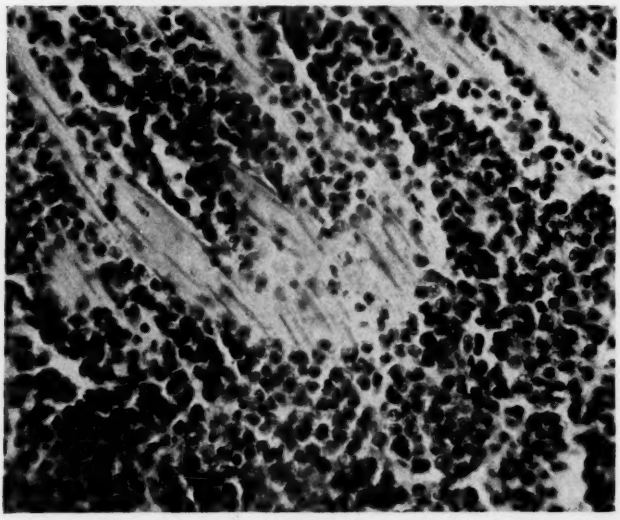

FIG. 11. Another view of the tissue at a distance from the ulcer, showing small round cell carcinoma. The undifferentiated type of cells and the mitotic figures indicate a high degree of malignancy. (X 400.) (Case 5.)

The patient died suddenly from a heart attack, three years and five months after operation. She had been shown at a meeting of the Richmond Academy of Medicine a short time before her death and was then apparently cured of

the malignancy. Unfortunately, there was no necropsy, but from the clinical history there were no symptoms of recurrence.

The majority of cancers of the stomach occur in the right half of the stomach, and in such instances anastomosis with the duodenum can usually be made. For eleven years I have used with much satisfaction a modification of the Billroth-I type of partial gastrectomy, which I have fully described elsewhere. In elderly patients a local anesthetic is indicated, and this technic is particularly suited to such an anesthetic. Even though there may be no free acid before operation, if the operation is successful the stomach may resume the secretion of acid. It is a well-known physiologic fact that the sensitiveness to the acid of the gastric juice increases from the duodenum downward, so that a recurrence of the secretion of acid may induce a jejunal ulcer. That this is not entirely theoretical is sustained by a case report by Dr. Fordyce B. St. John. The patient was operated upon for gastric cancer, the Billroth-II (Polya) type of operation being done, and later developed a jejunal ulcer and died.

While total gastrectomy has a very limited field, this field should doubtless be extended. In total gastrectomy drainage of the left hypochondriac region is indicated. In addition, I believe it is an improvement on the usual technic after selecting a long loop of jejunum and uniting the side of it to the stump of the esophagus, to make an end-to-side anastomosis of the duodenum to the right half of this loop instead of closing the stump of the duodenum. This anastomosis with an entero-enterostomy of the jejunum below the level of the transverse colon makes a three-point fixation of the jejunum, instead of a two-point fixation with the danger of volvulus. It also provides for the ready emptying of the duodenum.

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SPINAL ANESTHESIA
With a Note on Evipal

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IN 1933 the medical literature contained many articles about a new anesthetic, evipal. Weise, for example, stated that in 10,000 cases there was not a fatality from its use. Evipal is a white powder readily soluble in water but unstable in solution. It is furnished in 1 Gm. ampules. The powder is dissolved in 10 c.c. of distilled water just before using. The drug is rapidly decomposed by the liver; repeated doses fail to damage the normal liver, but its use is contraindicated by serious hepatic disease. It has no effect on the kidneys. When given intravenously during two minutes in sufficient dosage to produce narcosis, there is relaxation without preliminary excitement: with the completion of the injection full effect is obtained.

Morphine and the barbiturates are used preoperatively as with other anesthetics. I usually give the patient two nembutal capsules in a few ounces of tap water by rectum about an hour before operation, followed by morphine and atropine 15 minutes later.

The dose is 6 c.c. for every 100 pounds of body weight. The first 4 c.c. of the solution are injected in one minute, the remainder of the dose a little faster.

Anesthesia induced by evipal is deep and of short duration. Necessarily the selection of this anesthetic is determined by the type of operation to be performed: it should not be regarded as a universal anesthetic .

SPINAL ANESTHESIA

The history of spinal anesthesia dates from 1885 with the experiments of J. S. Corning, a neurologist of New York. Cocaine was the drug used and for this reason his method, notwithstanding several successful experiments, did not meet with general favor and was discontinued until 1898. In that

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year, Bier of Kiel, began experimenting with cocaine again, using himself, his assistant and six patients as subjects. He was successful in obtaining anesthesia to the umbilicus, but realized that cocaine anesthesia was too dangerous to be of practical use. However, he predicted that chemistry would produce a drug which could be used safely.

To Tait and Cagliere goes the credit for the first surgical operation in America under spinal anesthesia; but the distinction of perfecting the technic belongs to Jonnesco (1898). Since then spinal anesthesia has become popular.

Shortly after beginning the use of spinal anesthesia four years ago I had an unfortunate experience. The patient was a young negro woman with an ovarian cyst. I injected 200 mg. of novocain, placed her in the Trendelenburg position and started the operation. Almost immediately she began vomiting and sweating, her pulse became imperceptible and respiration slow and shallow. She was given ephedrine, caffeine and other stimulants but gradually ceased to breathe. For a while I practically discontinued giving spinal anesthetics. However, I have since resumed its use and now have a series of over 500 operations with no fatalities and very few untoward reactions. With my present knowledge I think this tragedy would have been prevented.

The indications for spinal anesthesia are well known.

The contraindications are:

1. Patients with systolic blood pressure below 90.
2. Patients with an almost complete suppression of urine.
3. Deformities of the spine in which lumbar puncture cannot be performed.
4. Infection at the site of puncture and diseases of the cerebrospinal system, such as meningitis or brain tumor.

Definite contraindications are therefore limited. In cases of a poor operative risk, spinal seems to be the anesthetic of choice. Spinal anesthesia allows sufficient time in prolonged operations and the operative work can be done painlessly and with almost no shock because the impulses are prevented from reaching the brain.

In our clinic we employ two preparations, novocain crystals and pantocain solution. When the anesthesia is to last not

longer than 45 minutes, novocain is used because it acts quicker and the effects wear away more rapidly. If the operation is expected to last one hour or more we use pantocain.

Pantocain is ten times as toxic as novocain but the effective dose is one-tenth that of novocain, so that its relative toxicity is the same. The ampules contain 2 c.c. of a 1 per cent solution of pantocain. The adult dose ranges from 1 to 2 c.c., depending chiefly on the duration of anesthesia desired and the weight of the patient. We usually employ from 1.0 to 1.75 c.c. averaging 1.5 c.c., and find this sufficient to induce anesthesia to the nipple line lasting from one and a half to two hours. The more I use spinal anesthesia the more I am convinced that there is a tendency to use excessive doses.

A possible drawback to pantocain is that it sometimes requires from 15 to 20 minutes to produce anesthesia. However, this slow absorption of the drug results in less reaction and may be of advantage. The solution has a specific gravity approximately that of the spinal fluid and so the operator need not deviate from his accustomed technic.

The preparation of the patient is important to secure a successful anesthesia and attention must be paid to small details. One hour previous to the operation the patient is given two nembutal capsules in solution by rectum and fifteen minutes later 1/6 or 1/4 grain of morphine is administered subcutaneously. The patient then comes to the operating room without fear and usually lies perfectly quiet, a distinct advantage as movement of the head induces nausea and vomiting. Many do not restrict the fluid intake but I prefer to give the patient nothing by mouth for two hours previous to the operation.

The technic of lumbar puncture has been fully described in many textbooks. It requires practice, however, to become expert. I usually select the third or fourth interspace for the point of injection, but it makes little difference.

It is important that the patient suffer no pain. This can be avoided in making the lumbar puncture, and the operation should never begin until anesthesia is complete. If we hurt the patient, even though the anesthesia is successful, he may still complain and be uncooperative.

The proper *type of needle* is essential in administering a successful anesthetic, and most failures can be attributed to one or both of two causes: (1) the use of a long beveled

needle, which permits only partial introduction of the solution into the canal, the remainder escaping into the tissues outside, and (2) movement of the patient after successful puncture may cause the point of the needle to shift, allowing the fluid to escape into the surrounding tissues. I use a 20 gage, flexible, rustless steel needle with a very short bevel. Just before doing the lumbar puncture, many operators advocate the use of ephedrine. I see no advantage in this.

The spinal fluid should drop freely from the needle and it must be clear before the anesthetic is injected.

If novocain crystals are used, about 2 c.c. of fluid are allowed to drip into the ampule containing the crystals, which rapidly dissolve. This solution is then mixed with an equal quantity of spinal fluid and injected. I never use more than 150 mg. of the drug. When using pantocain the 2 c.c. solution is aspirated into a 10 c.c. syringe and mixed with 2 c.c. of spinal fluid. Any desired dose may then be injected, each c.c. representing 50 mg. of the drug. The rate of injection should be approximately 0.5 c.c. per second.

The height of the anesthesia depends on the amount of the drug used, the dilution, and the force and speed with which it is injected into the spinal canal, and not on the interspace selected.

If one wishes a low anesthesia, very little fluid should be used in dissolving the crystals and in the secondary dilution, and the injection should be made very slowly with as little pressure as possible. The needle is rapidly withdrawn, and the patient placed immediately on his back for ten to twenty minutes. This is sufficient time for the drug to act, and then the patient may be placed in any desired position.

I never have the blood pressure taken during the operation, but do pay close attention to respiration, consciousness and the appearance of nausea of vomiting.

The earliest symptoms of the drug reaching the medulla is nausea. This occasionally occurs and may be followed by rather severe vomiting. At the first appearance of nausea, talk to the patient, insist on his taking regular, deep breaths, and assure him that he is all right and the nausea will pass away in a few minutes. If respiration does not improve, give him oxygen, and, if necessary, artificial respiration. If I had stopped the operation and given artificial respiration to the

young negro woman who died, I believe her life would have been saved. Keeping the patient comfortable, breathing deeply, talking, using oxygen and artificial respiration are far more important in combating untoward reactions than all the stimulants. If these symptoms do not occur within the first ten or fifteen minutes after injection, one may feel that the likelihood of a bad reaction has passed. I have seen the blood pressure drop to 30 mm., even with ephedrine, but it rapidly returns to normal, and as long as the patient is not nauseated, is conscious and breathing, the operator need have no fear so far as the spinal anesthetic is concerned.

After operation the patient is put in the reversed Trendelenburg position with low shock blocks for three to six hours and then kept flat in bed for twenty-four hours. One must remember that the extremities are paralyzed and the nurse must be careful in the use of hot-water bottles. I recently had a patient to suffer a rather severe burn of the foot following spinal anesthesia.

The anesthesia usually wears away in about one hour and a half when novocain crystals are used, but with pantocain the anesthesia, especially of the extremities, may persist for from two to four hours. It is advisable to tell the patient beforehand that the legs may feel paralyzed for several hours, so as to prevent undue worry.

I find no mention in the literature of a diminution in the output of urine following spinal anesthesia. However, I am confident that due to the lowered blood pressure there is an actual diminished output of urine during the first twelve hours. This seldom lasts longer than twenty-four hours when the urine output quickly returns to normal. It may be advisable to administer caffeine to these patients the night before the operation.

SUMMARY

1. The toxic effects of the anesthetic are greatly reduced. This is particularly advantageous in patients with metabolic disease, such as diabetes mellitus, and in those with liver and kidney disturbances, blood disorders and infarctions.

2. Shock is almost entirely avoided, because the conduction of impulses from the periphery to the brain is temporarily interrupted.

3. Relaxation of the abdominal muscles is more complete than with any other form of anesthesia. The intestines become contracted, peristalsis is arrested, and there is no necessity for forcibly packing away distended coils of bowel. For these reasons the surgeon can operate quickly, with greater ease, with less trauma, and with minimal dissemination of infected material.

4. Major surgery becomes possible in many cases where inhalation anesthesia is contraindicated.

5. In emergencies, spinal anesthesia enables the surgeon to proceed promptly without a trained anesthetist.

6. Electrical apparatus may be used without the danger of explosion.

7. Postoperative discomfort is lessened.

8. Patients are able to take liquids and carbohydrates before, during and after operation. The necessity for catheterization is infrequent.

9. Complications such as aspiration of mucus or infectious material are reduced.

10. Postoperative hernia is less frequent.

11. Postoperative ileus is rare.

12. The mortality is lowered.

13. From the patient's standpoint spinal anesthesia is usually more pleasant than inhalation anesthesia.

TETANUS AND ITS TREATMENT

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THAT the mortality in tetanus today (40-70 per cent) is only slightly less than during the pre-antitoxin era (68-84 per cent, Graves; 41-50 per cent, Hessert) demonstrates conclusively the necessity for a more thorough understanding, and a more rational consideration of both the prevention and treatment of this disease. Such a high mortality in an almost preventable disease can only be the result of failure on the part of both laity and medical profession to appreciate the seriousness of the disease and the importance of prophylactic measures.

Tetanus is a specific disease, produced by the absorption of a soluble toxin liberated by the bacillus tetani, resulting in a profound toxemia. This toxemia produces the characteristic muscle spasm, convulsions, and death.

The incidence of tetanus varies according to geographic location. It is more frequent in the South than in the North; however, there is slight seasonal variation with a higher incidence in summer. This certainly would be expected since puncture wounds produced by nails and splinters accounted for 50 per cent of the 813 cases reported by Graves. He also found a higher incidence in children and in males, probably because of their greater activity and exposure in summer. The incidence of tetanus in civil, industrial, and war injuries is of great interest. The general admission to Charity Hospital from 1840 to 1930 totaled 980,245 cases. The incidence of tetanus was 0.14 per cent. Wainwright collected 1,237,500 industrial injuries with a tetanus incidence of only 0.0008 per cent, and in the World War there was an incidence of 0.117 per cent in 2,053,142 wounded British soldiers. This extremely low incidence (0.0008 per cent) in industrial injuries demonstrates almost conclusively that tetanus is a preventable

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Read by Dr. Gage before the Sixth Annual Assembly of The Southeastern Surgical Congress, in Jacksonville, March 11, 12 and 13, 1935.

disease. The almost complete elimination of tetanus from the above collected series of industrial injuries is due to the intelligent application of prophylactic measures.

The etiology and especially the pathogenesis, both clinically and experimentally, have been of interest to a large number of investigators. Nicolaier, in 1885, injected dirt into animals, producing symptoms of tetanus. In the same year Kitasato isolated in pure culture the tetanus bacillus, and proved conclusively that this organism produced a soluble toxin which, when absorbed, produced all the clinical manifestations of tetanus in the experimental animal. In 1890 Kitasato and Behring produced antibodies in animals by the repeated injections of sublethal doses of tetanus toxin, and demonstrated the neutralizing power of these antibodies against the toxin. This important discovery was immediately utilized clinically as a prophylactic therapeutic measure.

The tetanus bacillus is widely distributed in nature. The majority of all animals harbor the bacillus in their gastro-intestinal tract. The excreta of these animals is used extensively as fertilizer. Therefore, highly cultivated land, as well as pastures, barnyards and streets contain a higher concentration of bacilli than barren land. Man also harbors the tetanus bacilli and spores in the gastro-intestinal tract as shown by Tenbroeck and Bauer. They found this organism in 34.7 per cent of 78 patients not having tetanus. Buzello and Rahmel found tetanus spores and bacilli in 50 normal individuals; 40 per cent had spores and 25 per cent spore bearing bacilli. This certainly verifies the commonly known fact that the tetanus bacillus is widely distributed in nature. All materials made from the hair of animals, especially wool, probably harbor the spores of tetanus bacilli.

The tetanus bacillus is an anaerobic, spore-bearing, saprophytic, toxin-forming organism capable of growth in the human body only under selective environmental conditions. The bacillus, when actively growing, produces a soluble toxin which, according to Ehrlich, is divisible into two specific types, namely, tetanospasm and tetanolysin, the former being responsible for the neuromuscular manifestations, the latter not entering into the production of tetanus. Another important contribution regarding the tetanus bacillus is that of Tulloch who, in 1917, isolated the tetanus bacillus from wounded

soldiers, and who classified the bacilli into four groups by agglutination with anti-bacterial serum. He subsequently demonstrated the mortality of each group to be the following: Group I—11 per cent; Group II—25 per vent; Group III—35 per cent; Group IV—0 per cent. He also noted that Group I was most frequent. These types remain true to form even after long cultivation. In view of Tulloch's findings, we should try and group our cases, because it may be a criterion in prognosis.

The development of tetanus in man is dependent upon several requisite factors, namely, the introduction of the tetanus bacillus or spores into the wound, devitalized tissue, the presence of pyogenic organisms, anaerobiosis, foreign bodies, and proliferation of the bacilli and toxin production. The introduction of the tetanus organisms into the body occurs through a break in the continuity of the skin, most frequently produced by puncture wounds (50 per cent, Graves). The organisms will not multiply and produce toxin in normal tissue due to its anaerobic characteristics. It has also been shown experimentally that toxin free spores or vegetative forms introduced into normal tissue are inert. However, if devitalization of tissue or secondary pyogenic infection occurs at the site of inoculation the bacilli will proliferate, liberate toxin and produce tetanus even as late as six weeks afterwards. Therefore, for tetanus to develop there must be present in a wound devitalized tissue, produced by the retention of infected foreign bodies, or suppuration, or both, the devitalized tissue acting as a culture medium and aiding in anaerobiosis. These factors promote and abet the proliferation of the tetanus bacilli that have contaminated the wound, with the liberation of toxin, and the production of toxemia. The tetanus bacillus, with rare exceptions, remains confined to the wound, liberating toxin which is absorbed by the lymphatics and blood stream. Once absorbed by the blood stream the toxin is confined to the circulating fluids for only a short period of time. In the experimental animal the toxin only remains in the blood for eight hours. The blood stream acts as a conveyor of the toxin to the neuromuscular end-plates of the motor nerves. The toxin is rapidly absorbed from the circulating blood stream by the end-plates of the motor nerves and transported to both the spinal cord and brain. When the toxin reaches

the spinal cord and brain it becomes inseparably fixed in the nerve tissue. The mode of fixation has never been satisfactorily explained. However, Almagia and Mendez concluded that lecithin and cholesterin were the preformed recipients which fixed the tetanus toxin in the central nervous system, and on this assumption, reported two cases successfully treated with injection of cholesterin. Huntington states "that fixation of toxin in nerve tissue is not prevented by extracting the nerve tissue with ether, and the 'magic words,' lipotropic fixation, do not apply."

After fixation of the toxin in the nerve tissue it is probably removed by catabolic reactions, and subsequently is excreted. This active autogenous detoxification depends entirely on the activities of metabolism. The mode of ascension in the peripheral motor nerves has never been definitely determined, but from experimental evidence the axis cylinder, epineural, and perineural lymphatics have been designated as the pathway in the nerve trunks (Marie and Morax; Meyer and Ransom; Field, Aschoff and Robertson).

The rate of ascension of the toxin in the peripheral motor nerves is rather rapid. We believe one of the factors which not only augments the rate of ascension but increases the amount of toxin transported to the central nervous system is the massaging effect on the peripheral nerves by the violent clonic exacerbations of the tonic muscle spasm. This massaging action is analogous to the so-called "secondary heart" or the normal physiologic action of the contracting skeletal muscles in "milking" the venous blood towards the heart. In our opinion this factor has never received sufficient emphasis. After the toxin has entered a peripheral nerve, it cannot be removed by any known method, but steadily progresses up the nerve and enters the cord and brain. It has been shown (Browning) that when an experimental animal has been so completely immunized by antitoxin that the blood and lymph contain sufficient antibodies to neutralize several times the lethal dose of toxin, nevertheless, the animal will develop tetanus if the toxin is injected into the sciatic nerve. This experimental observation proves that it is difficult to neutralize the toxin after it has left the blood stream and entered the peripheral nerve trunks. The peripheral nerve trunks can absorb and contain several times the lethal dose of toxin yet

the patient only shows mild symptoms, which become progressive as diffusion into cord and brain continues.

The lesions produced by the action of the toxin on the body tissues are slight and according to some observers are inconsequential and probably are not due directly to the toxin. Goldsmith and Flatau have described changes occurring in the anterior horn motor cells in experimental animals. These changes consist of swelling and disintegration of the Nissl bodies, with a concomitant enlargement of the cell and nucleus, the latter becoming less distinct. They have confirmed these findings in a patient dying of tetanus. On the other hand, Zimmerman, quoted by Huntington, states "that Nissl changes in tetanus are the result of exhaustion rather than the cause of any of the symptoms." We subscribe to Zimmerman's opinion, because Crile has conclusively demonstrated in both animal and man that identical neurocellular changes occur in exhaustion.

The symptoms of tetanus are dependent upon the virulence and number of organisms, the slow or rapid production of toxin, and the resistance of the individual. There is usually a history of some type of antecedent wound, puncture wounds being the most frequent, which the patient considers insignificant. Even after the onset of clinical symptoms the wound may appear inconsequential or apparently healed.

The clinical manifestations are most frequently pain in the muscles of mastication, followed by spasm of the masseter muscles. These manifestations occur first in the facial, cervical, and spinal muscles, and are thought to be due to the shortness of the nerve trunk by which the toxin travels. However, according to Sherrington, "the excitation of an agonist; e. g., flexor muscle, is regularly accompanied by inhibition of its antagonist, e. g., corresponding extensor, and that, therefore, normally both cannot be caused to contract reflexly. One may look upon this as due to the fact that between two antagonistically coordinated motor cells there is always a reciprocal inhibitory mechanism which so acts that when a cell is excited, the antagonistic cell is automatically inhibited." (Meyer and Gottlieb.) Regardless of the mechanism the masseters are the first to reveal spasm (tetanus descendens), which generally become progressive, and any attempt at facial expression results in the characteristic picture of "risus sardonicus." Spasm

of the masseters may last for several days before other muscles become involved; subsequently there is a gradual progression to all the muscles. Opisthotonos occurs when the muscles of the cervical region become spastic, and occasionally the head is drawn backward until it rests between the shoulders. The abdominal muscles are involved next, being manifested by a board-like rigidity. This rigidity interferes with the normal movement of the diaphragm resulting in varying degrees of anoxemia. The clonic exacerbations of the tonic muscular spasm are manifested by convulsive seizures. The convulsive seizures vary somewhat in their frequency, duration, and degree of intensity. Convulsions are usually precipitated by the slightest stimuli, as the motor cells and the reflex arcs are made very sensitive by action of the toxin. The convulsive seizures are so violent that cases have been reported in which fractures have occurred. During the convulsive seizure the patient may become cyanotic and respirations become very shallow and labored, or cease altogether, to be resumed after the seizure has subsided. The diaphragm and the laryngeal muscles finally become involved, resulting in asphyxia and death. From the repeated and violent clonic exacerbations of the tonic muscular spasms, varying and progressive degrees of exhaustion rapidly occur. Another deleterious effect of the convulsions is the massaging of the toxin up the peripheral nerves toward the central nervous system. Therefore, control of the convulsions will prevent exhaustion and decrease the absorption of toxin.

The presence of fever is variable, some patients dying with normal temperature, while others with hyperpyrexia recover. However, the majority will have an elevation of temperature ranging from 99 degrees to 105 degrees F. Miller and Rogers state that no definite conclusion can be drawn from the height of fever, although none of their cases with fever of 105 degrees or higher recovered. This has not been our experience in a small group of cases.

The pulse rate is usually accelerated, due to the toxemia and also the demands made on the heart by the violent convulsive seizures and the tonic spasm of the majority of muscles.

There occasionally occurs involuntary escape of urine and feces; however, retention of urine and feces is not uncommon.

Death occurs during one of the convulsive seizures due to spasm of the diaphragm and closure of the epiglottis from involvement of the pharyngo-laryngeal muscles resulting in asphyxia.

The prognosis of tetanus is dependent upon several factors, the incubation period, virulence of the organisms· the number and severity of the convulsive seizures, and the time active treatment is instituted. The duration of incubation period has for a long time been of definite prognostic value. The shorter the incubation period the higher the mortality, and the longer the incubation period the lower the mortality. Calvin states that even the introduction of antitoxin has not modified this contention. He reports a mortality of 84 per cent in a series of cases in which the incubation period was less than 10 days, and a mortality of 25 per cent in which the incubation period was between 14 and 21 days. Graves, in a review of cases at Charity Hospital, found a mortality of 71.6 per cent when the incubation period was between 22 and 45 days. The majority of authors report somewhat similar results. The introduction of toxin-free spores or vegetative forms, and the presence of antibodies in the circulation of tetanus carriers have a direct bearing on the incubation period. It has been shown experimentally that toxin-free spores introduced into normal tissue are inert. However, if the site of inoculation is traumatized as late as six weeks, the devitalization of tissue ensues, or pyogenic organisms are injected at the site of inoculation, tetanus will develop (Francis). If the same phenomena occur in the human there will result a prolonged incubation period, whereas, if vegetative forms are implanted in the tissue and favorable environmental conditions are present, toxin production occurs rapidly, resulting in a short incubation period. Another factor which is questionable in the prolongation of the incubation period is the presence of antibodies in the circulation of an individual who is a tetanus carrier. Tenbroeck and Bauer have demonstrated the presence of antitoxin in the blood of normal individuals who harbor tetanus bacilli in their gastro-intestinal tract.

In our opinion the severity and frequency of convulsions have a definitive influence on the prognosis by increasing the amount of toxin entering the spinal cord. The absence of convulsions has a more favorable prognostic significance. There-

fore, in general, each convulsion increase the mortality 1 per cent. The time elapsing between the first symptom of tetanus and the institution of specific treatment is a factor of utmost importance regarding the outcome of tetanus. It has been shown experimentally that each hour after injection of toxin the amount of serum necessary to neutralize the toxin progressively increases. Therefore, the earlier the treatment with the specific serum is instituted, the more favorable is the prognosis. The use of prophylactic measures also influences the prognosis, in that patients who have received prophylactic treatment, both local and specific, will develop only mild forms of tetanus with a low mortality.

The treatment of tetanus should be considered under the captions: (1) prophylaxis; (2) local treatment of the wound; (3) neutralization of toxin by specific serum; (4) relief of muscular spasm and convulsions by sedation; and (5) the maintenance of nutrition.

The prophylactic treatment consists in applying the principles of prevention which, although well known, are too often neglected. These principles of prevention are, namely, debridement of all wounds, removal of all buried foreign bodies, prevention of pyogenic infection, and administration of adequate amounts of antitetanic serum. If the wound is extensive and becomes infected, then the administration of prophylactic serum at weekly intervals until all infection has subsided is imperative. Wainwright's collected series of 1,237,500 industrial injuries with an incidence of only 0.0008 per cent of tetanus, and Bruce's collected series of wounded British soldiers, 2,032,142, with an incidence of only 0.117 per cent, dramatically demonstrates the result of prophylaxis.

The local treatment of the wound, when tetanus has developed, is as important as the treatment of the disease itself. We are heartily in accord with Taylor, who states that all patients suffering from tetanus should be admitted to the hospital through the operating room. The wound should be debrided, preferably under general or regional anesthesia. Local analgesia by infiltration is contraindicated. All devitalized tissue and all foreign bodies should be removed and if pyogenic infection is not present, every effort should be used to prevent it. Prohibition of any form of cauterization of the wound cannot be too strongly emphasized, because it results in the

devitalization of tissue, thus defeating the purpose of debridement. If the wound has suppurated it should be opened widely and foreign bodies sought and, if found, removed. Even an apparently healed wound should be opened. Taylor has shown a rather high incidence of foreign bodies contained in healed wounds in patients with tetanus. The above procedures are not without danger because wide areas, or fresh, absorptive surfaces are exposed to the action of toxin as well as the breaking down of nature's barriers. Therefore, the first doses of antitoxin should be administered at the time of operation, while the patient is still under the influence of the anesthetic. Failure to appreciate the importance of this procedure may result in a fatality. These methods immediately remove the source of toxin production, prevent further absorption of toxin, and produce an aerobic state. The wounds secondarily infected with pyogenic organisms should be constantly observed and modern methods of treatment instituted. The importance of secondary infections cannot be overestimated, because the infection further devitalizes the tissue and promotes anaerobiosis. Not infrequently the secondary infection may be the cause of a profound toxemia and thus be responsible for the death of the patient instead of the tetanus.

The neutralization of the circulating toxin in the blood and lymph can only be accomplished by the administration of antitoxin. The antitoxin can only neutralize the toxin which is still confined to the circulating blood and lymph. Once the toxin has entered the peripheral nerves and has become fixed in the central nervous system, it must be detoxified by the patient as there is no known method by which the toxin can be separated from the nervous tissue. The earlier the institution of specific treatment, the greater the beneficial effect, because as hours intervene between the onset of symptoms and the institution of treatment the greater is the saturation of the peripheral nerves with toxin. The titer of toxin in the blood and lymph systems is at its highest at the beginning of the symptoms. Therefore, the antitoxin should be administered as soon as possible and in large doses, the amount depending upon the severity and duration of the symptoms (80 to 100,000 units in the adult). As has been demonstrated experimentally (Huntington), the amount of antitoxin necessary to neutralize a given amount of toxin in vitro is considerably less than that

required to neutralize the same amount of toxin in the experimental animal. Therefore, the time interval between the first symptoms and the administration of antitoxin is of utmost importance. We must not overlook the fact that the patient when first seen may have already absorbed a lethal dose of toxin from the blood stream by the peripheral nerves, when neutralization by antitoxin is impossible.

Daily injections of antitoxin should be given to maintain a high titer of antitoxin in the blood and lymph, the amount depending upon the severity of the symptoms, the extent of the wound, and the presence of suppuration.

There are four routes of administration of antitoxin: (1) intravenously; (2) intramuscularly; (3) intrathecally; and (4) subcutaneously. The time required for absorption into the circulation by the various routes are, respectively: immediate; 8 to 12 hours; a few hours; and 48 hours. Therefore, we favor and use the following method of the administration of antitoxin: 60,000 units intravenously, on admission; 40,000 intramuscularly, and then daily doses of 10,000 to 20,000, depending on the reaction of the patient to the first dose and the severity of the symptoms. Our opinion of the intrathecal route is succinctly expressed by Wainwright, who states that "the best way to increase the mortality of tetanus is to give the antitoxin intrathecally."

The prevention of convulsions and relaxation of the tonic muscular spasms is almost as important as the specific medication. As has been previously explained, each convulsion tends to increase the mortality. The convulsive seizures associated with tonic spasm produces a rapid and marked degree of exhaustion. Thus the importance of sedation in the prevention of these convulsive seizures is obvious, because it diminishes the massaging action of the muscles on the nerves during the clonic exacerbation of the tonic spasms, thus decreasing the amount of toxin expressed into the cord. The drugs used to promote sedation are innumerable, some being advocated for their supposedly neutralizing effect on the toxin. We opine the only type of sedation that is beneficial is that produced by a drug having a high anesthetic coefficient. Phenol, one of the first drugs used extensively, was proposed by Bacelli in 1893, and is still popular in some countries. However, Kitasato believes that it is ineffectual. Magnesium sulphate, introduced

in 1899 by Meltzner who advocated intrathecal injection in order to produce muscle relaxation, has met with varying degrees of success. The drug is very dangerous; respiratory failure is a frequent complication following its administration, and the results are by no means encouraging. The barbiturates are used rather extensively, but their action is of short duration, and of necessity, must be repeated frequently. They are excreted slowly and have an accumulative action which is undesirable. Curare and curarin have recently been revived by Florey and Harding. Curare was used in 1859 by Vella in the treatment of tetanus because of its paralyzing action on the neuromuscular motor plate. Florey and Harding state that "in rabbits that have received lethal doses of toxin with general convulsions, it was possible to abolish the violence of the convulsion but not to lessen their number." The two undesirable reactions resulting from curare are asphyxia from aspiration of foreign material and paralysis of the diaphragm, either of which may cause death. We believe that the drug is entirely too dangerous to be of practical value.

Avertin was first used in tetanus by Laewen of Germany in 1929, to control the convulsions and relieve muscle spasms. Avertin has all the requisite qualities of a safe anesthetic when properly administered. It is rapidly absorbed, detoxified in the liver, and excreted almost wholly by the kidneys, has no accumulative injurious effects, and produces anesthesia lasting from 3 to 5 hours. We have used avertin in all of our cases except when it was impossible to obtain it. We give 60 to 80 mg. of avertin per kilo of body weight, and repeat it when the patient shows signs of recovery from its influence. Subsequent doses need not be large. Avertin prevents convulsions, relieves muscle spasm, and facilitates nursing care by decreasing and abolishing the hypersensitiveness to stimuli.

Our plan of treatment is as follows: Under a general anesthesia, the wound is debrided, and a massive dose, 60,000-80,000 units, of antitetanic serum is administered intravenously and 20,000-40,000 units intramuscularly. Upon returning to the ward and when beginning to recover from the anesthesia, the patient is given the first dose of avertin. The avertin is then repeated as often as the patient shows sign of recovery from its effect, averaging 4-6 hours. The patient is kept practically anesthetized for varying periods of time; the longest

SUMMARY OF CASES

Case No.	Age	Sex	Race	Incubation Period in days	Type of Wound	Amount of Serum	Sedation	Duration in days.	Result
1	13 yrs.	F.	W.	14	Puncture wound, foot, splinter, secondarily infected.	150,000 U	Avertin	2	Arthus phenomenon. Died.
2	14 yrs.	F.	W.	9	Puncture wound of foot.	160,000 U	Avertin	10	Arthus phenomenon. Died.
3	8 yrs.	M.	W.	7	Puncture wound of leg.	40,000 U	Morphine	3	Inadequate treatment. Died.
4	19 yrs.	M.	W.	13	Puncture wound, foot, nail; foreign body present.	160,000 U	Avertin	22	Recovered
5	28 yrs.	F.	W.	5	Puncture wound, thumb, splinter, removed.	343,000 U	Avertin	30	Recovered
6	8 das.	M.	C.	8	Infected umbilical cord stump.	50,000 U	Luminal	14	Pneumonia Recovered
7	14 das.	M.	C.	10	Infected umbilical cord stump.	20,000 U	Luminal	10	Recovered
8	7 yrs.	F.	C.	11	Puncture wound, foot.	253,000 U	Avertin	16	Recovered
9	4 yrs.	M.	C.	18	Incised wound, foot.	157,000 U	Avertin	12	Recovered
10	8 yrs.	M.	W.	9	Puncture wound, thumb; foreign body present.	283,000 U	Avertin	21	Recovered
11	13 yrs.	M.	W.	11	Puncture wound, foot, splinter.	140,000 U	Luminal and Amytal	13	Recovered
12	15 yrs.	M.	W.	7	Puncture wound, foot, splinter.	273,00 U	Luminal and Amytal	21	Recovered
13	9 yrs.	F.	W.	3	Splinter in foot three weeks. Two days after operation had stiffness of masseters.	80,000 U	Avertin	21	Recovered
14	20 yrs.	F.	W.	5	Post-abortal.	120,000 U	Avertin	15	Recovered
15	9 yrs.	M.	W.	8	Abrasion.	120,000 U	Nembutal	12	Recovered

that we have kept a patient under the influence of avertin was 8 days. We are of the opinion that avertin is the best sedative in tetanus and recommend its use in all cases with the possible exception of tetanus neonatorum.

The maintenance of water balance and a high caloric, easily assimilated diet is essential in maintaining nutrition. The duodenal tube is inserted through the nose into the stomach when the patient is operated upon and while under the influence of a general anesthetic. Fluids and food are administered through the tube at regular intervals. Fluids are also administered by phleboclysis, in order that the patient should receive at least 3,000 to 4,000 c.c. of fluids per day. Transfusions are of benefit and are frequently resorted to in our clinic. Unmodified blood should be used, and we prefer the DeBakey-Gillentine method.

The bowels and bladder should not be neglected. Catheterization should be resorted to if retention occurs. Enemas become a necessity in order that avertin can be instilled into the rectum.

In the present series there were 15 cases, summarized in the table. There were 3 deaths in the series, a mortality of 20 per cent. Two patients died with symptoms of pulmonary infection, autopsy of one revealing an anaphylactic reaction occurring in the lungs (Arthus phenomenon). The other patient developed the same symptoms and possibly had the same condition. The third case was inadequately treated, as regards the wound, specific serum, and sedation. Eliminating these 3 cases, we have 12 cases, all acute and severe, without a death. We believe that by using the measures advocated above, the mortality can be considerably decreased.

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THE PRESENT STATUS OF WOUND TREATMENT

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The purpose of this paper is to emphasize a few well-known features in the treatment of wounds in general as we see them in a community hospital of one of our small town districts. Even in the rural sections of this machine age we encounter various types of wounds almost daily.

The symptoms of wounds are familiar to every medical man, the principal ones being pain, shock, loss of function, hemorrhage, and the physical appearance of the wound itself.

The cause of wounds is trauma, either direct or indirect, and it is very rare that any instrument causing a wound other than the surgeon's knife is sterile. The wounds we most frequently see are those from automobile and railroad accidents, sawmill and other industrial plant accidents, gunshot wounds, farm wounds, and knife wounds. They may range from slight abrasions of the skin to those of a most severe nature.

The first and most important consideration in the treatment of all wounds is hemorrhage and shock. Primary hemorrhage may be insignificant or an immediate cause of death, depending on the size of the vessel or vessels injured. It is well to bear in mind the color of the blood in profuse hemorrhage, as well as the character of the flow, in order to distinguish between arterial and venous bleeding. It is also well to remember the strong probability of an artery and its accompanying vein both being injured at the same time. The first aid treatment of hemorrhage is usually given by some layman or the family physician immediately following the injury, and consists of temporary stoppage of the bleeding by compresses and tourniquets made of anything convenient, applied between the wound and the heart in arterial hemorrhage, and in venous, distal to the wound. A tourniquet applied too long may endanger both life and limb due to the occlusion of both arterial and venous circulation, the sudden interruption to all trophic stimuli, and a starvation of the tissue below the tourniquet. Compresses immediately above the wound in case of an artery, below the wound in case of a vein, or in some instances

Read before the Sixth Annual Assembly of The Southeastern Surgical Congress, in Jacksonville, March 11, 12 and 13, 1935.

directly in the wound, is a safer procedure than a tourniquet, but more difficult to retain in position during transportation to a hospital. Occasionally it is necessary to close the wound by temporary sutures to arrest hemorrhage. Spontaneous arrest of hemorrhage sometimes occurs, and such an arrested hemorrhage must be carefully watched until permanent relief is available.

My experience in dealing with wounds incurred in rural sections leads me to suspect that the treatment of shock has not been given sufficient emphasis. The majority of such patients I see have been brought to the hospital in the back seat of a car, on a truck or in some other vehicle in a very uncomfortable position. Regardless of the extent of the injury, the weather, or what-not, it seems to be uppermost in the minds of all lay people and some doctors that these patients must be rushed to a hospital immediately, and we surgeons who receive them are often too anxious to clean up the wound or get rid of a mangled limb. Such procedures add additional shock to a wounded person, already in a bad condition, and, in my opinion, are responsible for more deaths in postoperative wounded patients than all other causes combined. Therefore one must first treat the shock, which is secondary to and coincident with stopping hemorrhage.

Perhaps the best definition of shock is that given by Crile who says, "shock is the depression of the vital powers arising from an overstimulation of the nerve centers followed by an exhaustion or inhibition of the vasomotor mechanism," all of which may be brought about by trauma producing violent impressions on the sensory nerve fibers which are conveyed to the nerve centers. The first principle, then, in the treatment of shock is complete rest for the patient, especially the part affected. Administer morphine in sufficient quantities to relieve the patient of pain, thus preventing further shock.

Just a word about stimulants in the treatment of shock. Strychnine has been and is considered a good stimulant. It stimulates the central nervous system, in fact, the entire system. According to Crile's definition of shock, we already have an over-stimulation of the nerve centers, so it stands to reason that further stimulation of these centers by strychnine is just what we do not want. "It goads the heart to increased action when that organ has not sufficient blood passing into it to con-

tract firmly and strongly." This additional shock caused by strychnine has been described as the "whip to the dying horse to make it pull." It is well to remember that strychnine and similar drugs should not be given to a wounded patient. Camphorated oil was used during the war as a powerful stimulant. I have had very little experience with it. Atropine, next to morphine, is one of the best drugs in the treatment of shock. Atropine is considered by Hare as a "sedative to the vagus; it acts upon the vasomotor system, combats the dilation of the blood vessels, and maintains vascular tone, opposes stagnation of blood in any vessel, and increases the amount of moving blood." It is especially valuable when the skin is very moist. It is probably better to give morphine and atropine in combination. In my opinion, these drugs are usually the only hypodermics necessary to administer. A cup of hot coffee, when the patient can retain it, is worth while. Hot liquids should be given by rectum, and it is very important that heat be applied to the body. In profound shock it may become necessary to bandage the limbs tightly. We usually do this by applying a layer of absorbent cotton over the limb, over which light bandages are applied, producing even pressure the full length of each limb. This has a tendency to inhibit the peripheral circulation and to keep the blood in the deeper organs where needed. Such treatment can be given anywhere.

The administration of tetanus antitoxin should never be forgotten, or better still, combined tetanus and gas bacillus antitoxin.

After the above treatment is carried out, the patient then should be taken to the hospital, if hospitalization is necessary, when he has fairly well reacted from the shock of the injury. Intravenous solutions of saline and glucose, and, where there is an excessive loss of blood, blood transfusions may be given before surgical interference. In other words, get the patient in the best possible condition and we can then proceed with such operative procedures as are necessary, depending on the nature and extent of the wound.

In most wounds local or block anesthesia can be and should be used, because it is the safest and tends to prevent further shock to a patient already depressed.

In the treatment of the wound itself there are a number of methods recommended especially regarding the antiseptic

used. We all have our hobbies in this respect. Morehead, who in all probability has one of the largest traumatic surgical practices in the country, uses iodine as the antiseptic of choice. When using iodine it is best to dry-shave and cleanse the skin with benzine, followed by the application of a 5 per cent tincture. Soap and water is, indeed, a splendid cleansing agent, but iodine penetrates more readily when applied on a dry surface. I appreciate the fact that there is considerable opposition by some good men to the use of iodine. Some feel that while iodine is a good antiseptic, it also injures the vitality of the tissue and, therefore, does about as much harm as good. I believe, however, iodine will continue to be the antiseptic of choice in fresh wounds. It must be remembered that in the treatment of all wounds no antiseptic really sterilizes the wound, and this is especially true in lacerated or jagged wounds.

After cleansing the skin as thoroughly as possible, the next step is the careful excision of all traumatized tissue. Debridement of wounds apparently was first suggested by Larrey, in 1814, during the Peninsula Campaign. One hundred and one years later Gray, of the British Army, and LeMaitre with the French, began doing more radical debridement around the channel of the wound, in addition to removing any foreign bodies. The technic of debridement should consist of a thorough excision of all traumatized skin, fat, fascia, and muscle, including a thorough search for concealed tracts, and the removal of all foreign bodies. The dissection should be done with very sharp and smooth instruments, and the operator should bear in mind the importance of preserving uninjured the nerves and blood vessels, most of which lie immediately beneath the fascia. Even retraction should be made so as to expose all bleeding vessels, which should be ligated or repaired well above any trauma or injury to the vessels. Bleeding vessels should be tied with the smallest catgut necessary to hold them. (For small ones we use No. 00 plain catgut, and chromicized for the larger.) Catgut is supposed to be sterile, and is as a rule, but it is advisable to leave as little excess foreign material in the wound as possible.

After a thorough mechanical cleansing of the wound and ligation of all blood vessels, the area should be flooded with ether and irrigated thoroughly with saline solution as a fur-

ther cleansing procedure, after which a clean set of instruments and gloves are put in use and all tendons and fascia sutured carefully, with fine chromic catgut. All dead space is obliterated. The skin is brought together with dermal or very fine silkworm sutures. One should avoid undue tension and see to it that the wound is dressed with even pressure.

After closure of the wound it is important that the patient be observed closely for the first forty-eight hours as some of these wounds will become infected, in which event all sutures must be removed and the usual treatment of infected wounds instituted.

Wounds that become infected following the above procedure, or that are already infected because of delayed treatment, should be left open and continuously irrigated with a mild antiseptic solution. The most popular antiseptic has been Dakin's, but others may be substituted, the main point being its continuous application. The length of time this treatment should be continued varies with the case. When bacteria are no longer present it is customary to perform a closure of the wound. There is one point in the treatment of infected wounds that I strongly emphasize; do not break down any healthy granulations as they appear. All of us have seen patients with infected wounds, as, for instance, those following the drainage of an appendiceal abscess, have rigors as the result of opening up small capillaries and allowing an absorption of infected material into the blood stream. We must be careful not to over-treat wounds.

In this connection I wish to call to your attention the bacterial destructive agent, or bacteriophage, as described by d'Herelle, formerly of Egypt, but now at Yale University. One of the most favorable reports of its use is by Albee, who found that bacteriophage occurred spontaneously in 94 per cent of cases of osteomyelitis treated by the Orr method, and who attributes the splendid results of Orr in his treatment of osteomyelitis to the bacteriophage. In the light of these observations it is reasonable to expect definite progress in the treatment of infected wounds by bacteriophage, especially when our laboratories become more proficient in the growing and isolation of races of the phage specific for each bacterium.

In the treatment of wounds I like to emphasize a feature already mentioned, and that is blood transfusions. Where a patient has lost a great deal of blood there is no other remedy as valuable as a blood transfusion, and I prefer to use the direct method. My experience with this method leads me to believe that when the patient's blood is correctly matched and typed reactions are very rare. Not only is blood transfusion valuable where the patient is anemic, but it also gives excellent results in prolonged cases of infected wounds where septic symptoms have developed. In such cases small transfusions frequently administered are more efficient than larger ones less frequently given.

Finally, the patient's general condition should be considered and the necessity of employing every means possible to raise the natural health forces, such as plenty of good, nourishing food and lots of sunshine. Where sunshine is not available the quartz lamp is a good substitute and a great aid in building up the patient's general resistance. It is also advisable to remove all foci of infection, as in the treatment of any other condition.

Conclusions

1. It is necessary to treat shock coincident with hemorrhage, if there be a hemorrhage, and not to add additional shock to a patient already exhausted by rushing him to a hospital or some other permanent place of treatment in a haphazard way.
2. Iodine is probably the best antiseptic we have for the treatment of fresh wounds.
3. Jagged or lacerated wounds should be treated by debridement before suturing instead of treating them only by antiseptics.
4. Blood transfusions should be given in cases where excessive loss of blood has occurred and where septic symptoms have developed in infected wounds.
5. Bacteriophage treatment of infected wounds may become the treatment of choice.
6. It is important to treat the patient's general condition and do everything possible to build up natural health resources.

THE DIAGNOSIS OF INTRACRANIAL TUMORS

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ONE of the chief factors in the diagnosis of intracranial tumors is to convince the profession that such lesions exist in far greater numbers than has heretofore been thought, and, furthermore, that surgery offers complete cure to a great many and alleviation of symptoms to those who can not be permanently cured. My reason for making this statement is not to be construed as a criticism of the diagnostic acumen of our colleagues in other lines of endeavor, but rather to point out that there is apparently a miscomprehension in regard to both the incidence and operative mortality of brain tumors. This is possibly due to the failure of those of us who are engaged in neurosurgery properly to disseminate the advances made in diagnosis and treatment in the recent past

In the foreword to his excellent volume on Tumors of the Brain, Ernest Sachs states that "since attention has been focused on neurosurgery in this country, the neurosurgical material in every surgical clinic, which has a neurosurgeon on its staff, has increased amazingly and now constitutes a considerable part of the surgical material."

In the Cushing series of verified tumors, comprising a thirty-year period prior to 1928, 2,716 cases came to operation. But, during the last six years of this period there were 1,427 cases, 38 more than in the preceding twenty-four years.

Those of us who live in the South are familiar with the oft-repeated and time-worn statement that negroes do not have brain tumors. There is no biologic basis for this statement, its only foundation resting upon the fact that heretofore we have not diagnosed them.

In the consideration of intracranial neoplasms from a diagnostic standpoint it is necessary, in order to obtain a proper perspective, to discuss briefly certain anatomic and pathologic phases. These tumors may in general be described as arising from:

Read before the Sixth Annual Assembly of The Southeastern Surgical Congress, in Jacksonville, March 11, 12 and 13, 1935.

1. The brain substance itself, the glioma group.
2. The coverings of the brain, the meningiomas, blood vessel tumors and the questionable sarcomas.
3. The pituitary gland and its stalk.
4. The pineal gland, the pinealomas, and
5. From certain cranial nerves, notably the eighth—the acoustic neurinomas.

Those tumors having their origin from the brain substance do not produce the definite symptomatic pattern seen in the other groups. The reason for this is apparent when one considers the various sites from which they originate. These tumors vary from the practically benign astrocytoma to the highly malignant spongioblastoma multiforme. There is great variance also in their rate of growth—from a few weeks to many years. The degree of malignancy can be determined by the duration of the patient's symptoms.

Representative of the tumors arising from the coverings of the brain are the meningiomas. These tumors have their origin from fairly constant points and oftener than not produce a clear-cut syndrome depending on their location. They originate from the attachment of the dura to the crista galli producing the so-called olfactory groove tumors on the floor of the frontal fossas. While its point of origin is always unilateral this tumor not infrequently extends under or through the falx cerebri to produce bilateral symptoms. Its rate of growth is so slow that it often attains the size of an orange or small grapefruit before producing signs of increased intracranial pressure. The syndrome produced is ipsolateral anosmia and primary optic atrophy with papilledema of the contralateral disc. When of sufficient size to damage the frontal lobes, symptoms referable to personality and memory may result.

Another frequent site of origin of this group of tumors is from the sphenoid ridge where the dura has its attachment. Arising at any point on the ridge, as they slowly enlarge, these tumors can encroach on the structures at the sella turcica medially, the frontal lobe and olfactory nerve anteriorly and the temporal lobe posteriorly. When far enough lateral, on the greater wing of the sphenoid, the syndrome is not as distinctive as when on the lesser wing. In the latter instance one usually encounters ipsolateral optic atrophy and homonymous hemianopsia. The greater wing group does not, as a rule, im-

pinge on the optic nerve and, therefore defects of the visual field are not produced. However, papilledema, secondary atrophy and anosmia are usual. In this location occasionally unilateral exophthalmos is seen. In this event there is primary atrophy of the nerve of the affected side, progressing eventually to complete blindness in this eye with choking of the contralateral disc.

Still another favored site of origin of this same type of tumor is at the sella turcica just anterior to the optic chiasm. In this instance the syndrome is a primary bilateral optic atrophy and bitemporal hemianopsia without x-ray evidence of alteration in the size of the sella as is seen in enlargement of the pituitary gland itself. This tumor will eventually produce total blindness unless it is removed.

The pituitary tumors produce probably the most definite syndrome of any of the other groups. These may be placed in two classifications, the tumors of the gland itself and the craniopharyngeal pouch cyst. The former are true neoplasms while the latter are the result of a developmental anomaly in which there has been faulty fusion between the pars nervosa and pars buccalis of the gland. The adenomas are recognized by the same eye signs just described for the suprasellar meningiomas, but there will be x-ray evidence of enlargement of the sella in this instance. In addition one may observe varying degrees of endocrine disturbance. The craniopharyngiomas are usually recognized by a characteristic roentgenographic shadow of suprasellar calcification.

The pineal tumors are exceedingly rare. The main diagnostic signs are an early and great increase in intracranial pressure due to occlusion of the aqueduct, and a paralysis of upward gaze. Also one obtains x-ray evidence of the presence of pinealomas due to excessive calcification here.

Tumors of the cranial nerves produce definite signs referable to the distribution of the particular nerve involved. One should remember that the early signs are irritative, slowly progressing to complete paralysis as the nerve is pressed upon. Signs of an increase in intracranial pressure may be late. Most commonly the eighth nerve is the one affected, though there are instances in which the second, fifth and ninth may be the sites of origin. In the acoustic neurinoma group the earliest signs will always be referable to the eighth nerve, but as the

growth slowly expands many of its neighboring nerves, as well as the cerebellum, become encroached upon, thereby producing many additional signs. This is usually a tumor of early middle age and is quite slow in its progress.

As in the proper diagnosis of other disease processes a properly taken history is the sine qua non in the diagnosis of intracranial tumors. The functions of the brain are the reception, initiation and transmission of impulses of various sorts, as well as the fabrication of concepts—the origination of psychic processes, and the storing away of experience—memory. Every tumor of the brain begins, of course, as a very small lesion, gradually or rapidly, according to the nature of the lesion, expanding in size. The symptoms that are produced are therefore first of an irritative nature, progressing to a complete paralysis of the functions of the particular area of the brain involved. In addition to these localizing symptoms thus produced, one also observes neighborhood symptoms due to edema of brain tissue immediately adjacent, and distant symptoms due to changes in remote areas dependent on interference by pressure with the blood supply or cerebrospinal fluid circulation. It thus becomes obvious that a definite chronologic sequence of events obtains in the symptomatology of brain tumors, and this fact is of the greatest importance in the carefully elicited history. Facts of trivial import to the patient, such as tinnitus and diminution of hearing in the same ear, only noticeable perhaps in the use of the telephone with that ear, may be of extreme importance in a neurologic history. It is apropos, I think, to mention at least some of the symptoms one is likely to encounter.

1. *Psychic Changes.* Many patients with brain tumor have been diagnosed as mental cases and confined in institutions for such cases, because the psychotic symptoms were conspicuous and the obvious signs of increased intracranial pressure had not yet developed.

Many a case of so-called general paresis has been the unfortunate victim of an unrecognized brain tumor. In all fairness, and in the light of our present knowledge as to the incidence of tumor, it is not too extreme to insist that before a patient is consigned to an institution, that at least consideration be given to the possibility of the presence of brain tumor, and the proper diagnostic procedures be instituted to elimi-

nate this possibility. When present the mental symptoms will range from simple deterioration to profound dementia. At times there is seen a euphoric state in which the patient is not in the least concerned about his grave predicament and even "wise cracks" when told that he is confronted with a serious surgical procedure.

2. *Convulsive Seizures:* When a patient is presented with the complaint of generalized convulsive seizures it should be borne in mind that in idiopathic epilepsy symptoms develop in over 50 per cent of victims before the age of 15, in over 85 per cent before the age of 30, and in over 90 per cent before the age of 40 (Bailey). It has been observed that in young adults, in whom the presence of syphilis has been disproved, tumor is the most frequent cause of convulsions (Sachs). Beyond the age of 35 or 40, the occurrence of convulsions has an increasingly significant importance. While syphilis, arteriosclerosis and renal disease are not infrequent etiologic factors these are as a rule readily demonstrated. From the fourth decade of life on, intracranial neoplasms are the most frequent factors in the causation of general convulsive seizures.

3. *Visual Disturbances:* These vary from a diminution in acuity to complete loss. Unfortunately, too many cases are referred for study in the latter state. Double vision is a common symptom. This has no localizing significance, but is due to the sixth nerve in its long course from just beneath the pons and across its surface to the optic foramen, being compressed against the basal structures by the general increase in intracranial pressure.

A patient suffering from brain tumor may have a steadily progressive loss of vision without the other two familiar signs of increase in intracranial pressure, namely, headache and vomiting. I know of a man who, because of visual failure alone, unaccompanied by other signs, went for three years from one physician to another before his eye grounds were examined and visual fields taken. When this was done there was found abundant evidence of pressure at the optic chiasm. He was operated upon and a pituitary tumor verified and removed. Complete restoration of function followed—but what about the three years of enforced idleness, as well as the

threat to permanent loss of his vision, that this patient was subjected to because of the fact that the visual disturbance complained of was not earlier recognized as a symptom of tumor?

4. *Tinnitus and Diminution in Hearing:* These are the first symptoms of tumors affecting the acoustic nerve. They may precede for years the neighborhood symptoms due to pressure on the adjacent seventh to twelfth cranial nerves and the cerebellar pontine pathways. In the latter event ataxic symptoms are produced and combine to develop the typical syndrome of the acoustic neurinoma already described.

5. *Unilateral Facial Weakness:* When only the lower segment is involved this is a most valuable sign. It may or may not be accompanied by a paresis of the arm and leg of the same side, depending on the extent of the lesion. The facial weakness is sometimes so slight as to be brought out only on emotional stimulation.

6. *Disturbance in Gait:* Many neurologic conditions as well as those related to other systems produce a staggering gait. However, it must not be overlooked that this is a symptom of importance when the cerebellum or certain of its pathways are interfered with by pressure from tumor formation. Accompanying symptoms may be ataxia, adiadochokinesia and hypotonia. Usually cerebellar tumors produce headache early in the course of their development due to the internal hydrocephalus resulting from occlusion of the aqueduct, fourth ventricle or the foramen of Magendie.

7. *Headache:* I have studiously avoided the mention of headache until now, because so often tumors may reach considerable size without the production of headache. This is particularly true of frontal lobe tumors where, due to their position, the cerebrospinal fluid circulation is not interfered with. The time has long since passed when one should not suspect the presence of brain tumor because of the absence of the familiar triad of symptoms of increased intracranial pressure, namely, headache, vomiting and dizziness. These are of importance when present, but in many instances the disease is far advanced before they make their appearance. Persistent headache and vomiting, the latter particularly in children, demands the most painstaking investigation.

A carefully taken neurologic history is time-consuming, but unless the physician is prepared to devote much thought and time to it, many failures in diagnosis will result.

The examination is no less arduous than the history taking. One should have a definite system, and should follow it precisely in every case. The examination is important not only because of its relationship to the neurologic problem involved, but because also of its bearing on the ability of the patient to withstand a major surgical procedure.

The most important features of the neurologic study are:

1. Eye grounds.
2. Visual fields.
3. X-ray examination of the skull, and
4. Ventriculographic studies.

Eye Grounds: Neglect of an ophthalmoscopic examination of the fundus oculi may be the cause of a tumor being overlooked until vision is irreparably damaged. The ophthalmoscope is as important in neurologic diagnosis as is the stethoscope in the detection of disorders of the heart and lungs. When choked discs are present it is presumptive evidence of the present of tumor.

Atrophy of the optic nerve, manifested by pallor of the disc, is of almost equal importance as the finding of papilledema. It always means pressure on the fibers of the optic tract at some point and demands adequate investigation.

Visual Fields: It is my opinion that examination of the visual fields is perhaps the most important single procedure in the clinical investigation of tumor suspects. Not infrequently a localizing diagnosis is made by this means alone. It is now generally agreed, from the standpoint of the neurologist at least, that the screen method of visual field examination is far superior to the perimeter, in that defects can be picked up weeks or months earlier than with the older method (Walker), thereby being the means of conserving vision which might otherwise be irreparably damaged. As much depends on the result of this observation it is obvious that meticulous care is essential in its performance.

X-ray Examination of the skull is indispensable. Perhaps 35 per cent of all tumors can be diagnosed by this alone. The

characteristic calcification of certain tumors and the proliferation and erosion of bone produced by the superficially placed meningiomas are almost pathognomonic. The visual disturbances described above, together with changes in the sella shown by x-ray films leave no doubt as to the presence and localization of the lesion. Radiographic studies frequently give positive evidence of a general increase in intracranial pressure by the now familiar picture of convolutional atrophy.

Ventriculography, as you know, consists in replacing the ventricular fluid by air followed by x-ray studies of the contour of the ventricles. Dandy, who originated the method, has stated repeatedly that, if properly done, it is without danger. On the other hand Grand in Philadelphia has statistical evidence that the procedure has a mortality rate of slightly less than 9 per cent. It is difficult to reconcile these statements. My own experience in a series of approximately 40 cases has, to date, been without untoward symptoms. My feeling is that ventriculography should be used only after careful and repeated neurologic examination has failed to give definite localization in cases where the presence of tumor is suspected—or when, in the observer's mind, the localization is not definite. Certainly the risk is justified by the information obtained when the injection is done under the proper conditions and by one familiar with all the possibilities.

Often after the most careful history has been taken and equally careful examination has been done, the localization of a tumor is still most elusive. In that event one can only rely on the basis of reasonable probability, based on the sum total of one's knowledge of neurology and common sense.

In the words of Foster Kennedy:

"He who cares for patients suffering from brain tumor must bring to his problem much thought and stout action. There is need also of a formidable optimism, for the dice of the gods are loaded."

Medical Arts Building.

THE SURGICAL TREATMENT OF ANGINA PECTORIS

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CORONARY thrombosis may be recognized by the agonizing pain produced, which is prolonged and continuous, pain which does not yield to rest or to the nitrites but which may be relieved by large doses of morphine. This pain of coronary occlusion arises from the resulting ischemia of the heart muscle.

Relative ischemia of the heart muscle from a temporary insufficiency of the coronary flow is considered to cause the paroxysmal pain of angina pectoris. While in most cases coronary disease sufficient to interfere with the blood supply of the myocardium will be found, in many cases of coronary disease discovered postmortem there is no history of pain, and the anginal syndrome is rare in negroes. Coronary disease is not therefore the sole factor. Angina pectoris results from a transitory functional disturbance. It seems to be a spasmodic disease occurring in patients who exhibit what Houston has called " the spasmogenic aptitude."

Statistical studies have shown that angina affects four men for every woman, and that the average life of an individual suffering from angina pectoris is five years following the initial attack. In an analysis of a group of 100 cases of angina pectoris treated medically, Green and Burton found that the immediate cause of death was some unusual activity which could have been avoided. When no organic lesion of the heart is present, however, the patient may live for many years with only slightly restricted activity and at length die a noncardiac death.

The diagnosis of angina pectoris in its early stages must often rest solely upon the one symptom of substernal pain following effort. Physical examination of the heart is negative in approximately one fourth of the anginal patients. In such cases the history becomes of major importance: painstaking questioning is often necessary.

Read before the Fifth District Medical Society of Georgia, in Atlanta, March 29, 1935.

"Anger, love, fear, hate and surprise more strikingly influence the heart rhythm than any physical factor that we know of," says Harlow Brooks in *Time,* and precipitate angina. He adds that "relaxation, music, diversion, congenial conversation and sunshine are good for heart cases and prevent anginal attacks."

Until recent years angina pectoris was considered entirely a medical problem. The first operation for the relief of anginal pain was performed in 1916 by Jonnesco, who removed

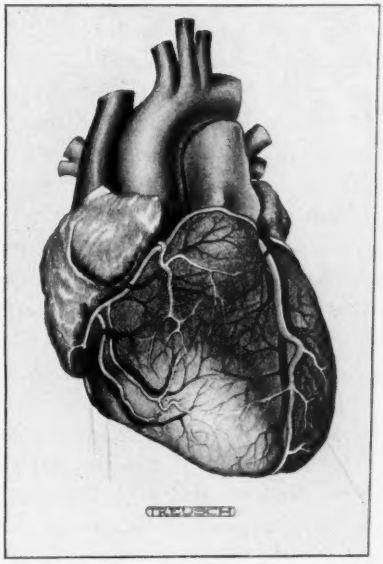

FIG. 1. Drawing showing infarction of the greater part of the right ventricle, the result of coronary thrombosis.

the superior cervical sympathetic ganglion, in an attempt to interrupt the pathways of cardiac pain. The results of this operation and its various modifications have been partially successful in about two thirds of the patients operated upon: the immediate mortality has been almost 10 per cent. Paravertebral alcohol injection of the upper thoracic sympathetic ganglion has failed to give satisfactory results in approximately 25 per cent of cases. Because of its dangers it is a method which should never be put into general use.

A more fundamental approach to the problem was begun twelve years ago when Willius, Boothby and Wilson wrote of

"the increase in the rate of blood flow in hyperthyroidism, and the resultant increase in the work of the heart, this increase being relatively in proportion to the increase in the basal metabolic rate." About the same time Boas stated that in thyrotoxicosis the volume output from the heart might be 25 to 60 per cent above normal. Blumgart, Weiss and others corroborated these observations and determined also that, when the metabolic rate is lowered, as in myxedema, the blood velocity is correspondingly retarded.

Working at the problem from another angle, Means, White and Krantz found that when the basal metabolic rate of an anginal victim was elevated by thyroid extract, there was a marked increase in the severity of the attacks. Conversely, Haines and Kepler have reported thirty-three cases of angina associated with hyperthyroidism with relief in each case following subtotal thyroidectomy. Others have reported similar though smaller series.

In uncomplicated cases of angina pectoris the heart is embarrassed by the partial ischemia of its muscle, that is, the heart is unable to meet the demands placed upon it by normal metabolism. Since the work the heart is called upon to do is in proportion to the velocity of the blood flow and this is retarded in hypothyroidism, and since increasing the normal metabolic rate of an anginal patient makes his trouble worse and lowering the elevated rate of an anginal patient makes his trouble better, it followed that the lowering of a normal metabolic rate, that is, the artificial induction of myxedema, should benefit angina pectoris.

Reasoning along this line led Blumgart, Levine and Berlin to perform subtotal thyroidectomy in a case of severe angina pectoris without hyperthyroidism in an attempt to lower the metabolic rate. The patient was temporarily relieved of his anginal seizures, but in a short time metabolism was restored to normal and the attacks recurred. Then they concluded that nothing short of total ablation of the normal thyroid gland would be of lasting benefit in cases of heart disease. This operation was undertaken on Dec. 14, 1932, at the Peter Bent Brigham Hospital in Boston: it has been repeated in various clinics all over the country since that time.

Local anesthesia is best, though anginal patients as a rule stand gas or ether well. There is the distinct advantage in the

use of a local anesthetic that the patient is awake and can talk to the operator so that a change in voice will give warning if the recurrent laryngeal nerve is endangered. It is my custom in all thyroid operations to give 3 grains of sodium amytal the night before and again two hours prior to the operation, and 1/3 grain of pantopon with 1/200 grain of scopolamine an hour beforehand. Most of my patients are drowsy but will talk as desired: rarely is a general anesthetic required. The average patient is able to leave the hospital in eight or ten days.

In the usual subtotal thyroidectomy the surgeon remains within the gland capsule, avoiding the parathyroid bodies and the recurrent nerve. In total thyroidectomy every vestige of gland substance must be removed, so the operator must come close to the parathyroids and the nerves. The chief complications of the complete operation are parathyroid tetany and paralysis of the vocal cords through injury to the nerve. Cutler has reported four cases of nerve injury and four cases of mild tetany in his twenty-nine cases. His immediate mortality was two (6.8 per cent), both patients dying of coronary occlusion within 48 hours of the operation. Mixter, Blumgart and Berlin have reported twenty-three cases without a death.

I have performed total thyroidectomy on ten patients with no operative mortality: two operations were for angina pectoris, three for congestive heart failure and five for carcinoma of the gland. Unilateral cord paralysis and parathyroid tetany followed one extensive dissection for carcinoma. The patient, however, is still living after twelve months with no recurrence and she is content to whisper and to take calcium gluconate three times a day.

I have the impression that the relief obtained from total thyroidectomy in cases of angina pectoris cannot be measured entirely in terms of the basal metabolic rate. The determination of the metabolic rate cannot always be relied upon as an index to the amount of damage hyperthyroidism has done the heart or to the relief to be obtained by surgically induced hypothyroidism. In both instances the clinical picture as a whole must be carefully studied, and the close cooperation of the internist with the surgeon both before and after operation is essential. On the other hand, the Boston observers report that their patients who showed no improvement were those

with a low preoperative basal metabolic rate: they consider such a rate a contraindication to operation.

It is unnecessary to keep the patient in a state of severe myxedema. Small doses of thyroid extract may be given to regulate the metabolism, being careful not to raise it to normal again. It has been found that one tenth to one half a grain daily is sufficient to modify the unpleasant symptoms of myxedema: one fourth grain is the average dose required.

The results following total thyroidectomy for angina necessarily depend, too, upon the degree of pre-existing myocardial damage and upon the cooperation of the patient. There is no

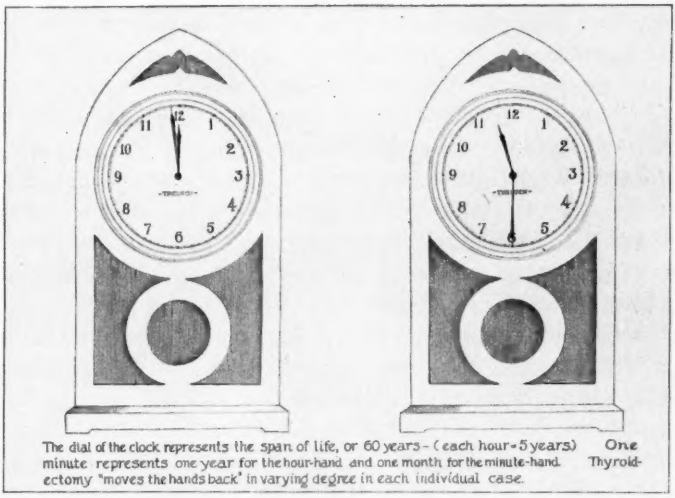

FIG. 2

reason to believe that total thyroidectomy greatly retards the progress of the underlying pathologic process in the coronary arteries. The thyroidectomy simply sets back the hands of the clock. The duration of the improvement will depend largely upon the patient's personal habits and the care he takes of himself. It is too early still to say whether these patients, as a group, will live longer than those who have the benefit of medical treatment only.

We may liken the patient with angina pectoris unto an old and wornout horse pulling a heavily loaded cart up a steep hill with the odds heavily against his making the grade: if

then suddenly the driver dumps a large part of the load to make it possible for the old horse to top the hill, that would correspond to total thyroidectomy. But the horse would still be old.

Irrespective of the scientific data accumulated and the differences of opinion for and against the operation, "total thyroidectomy offers to patients with angina pectoris a hope heretofore wanting."

The great difficulty in handling these anginal patients after operation is that many of them cannot understand that the operation has not cured the fundamental cause of the angina, and they insist upon resuming business activities too early and refuse to take proper care of themselves. Increased activity will in some cases cause a return of modified symptoms. However, if the patient will face his limitations, accept them and then live within them, there is no reason he should not lead a contented life and in many cases, a reasonably useful life. The problem of resuming work depends not only upon the condition of the patient, but upon the character of his occupation. The resumption of work should involve relatively short hours, the avoidance of hurry and worry, a noon-time period of rest, long hours in bed at night and a long vacation twice a year. If the patients are made to understand in the beginning that the operation does not cure the trouble, but will probably give some relief, they are satisfied and grateful.

Report of Cases

Case 1. In January, 1934, Mr. B., a druggist, aged 44, began to have occasional attacks of pain and a feeling of pressure in the left chest, around the heart, with shortness of breath. His blood pressure was 170/96; the heart rate 85 to 90. On March 7, he had a definite attack of angina due to the shock of a sister-in-law's suicide. He suffered for three or four days, and was relieved by nitroglycerine and codeine. He experienced a severe attack on March 11 while at a wrestling match and was compelled to lie down. After several days he was again relieved by drugs. On March 15, an electrocardiogram "showed partial occlusion of coronary artery." The patient was kept in bed and given nitroglycerine. He became morose and despondent. He was allowed to sit up in April, then began to feel better and walked about the house. He had another severe attack on May 26, with pain radiating down the left arm. He was then kept continuously in bed on a liquid diet, as otherwise he would have mild attacks of angina.

In July he was brought to the hospital in an ambulance. He was a well-developed man, weighing 198 pounds. The heart was apparently normal in size, the sounds were normal, the rate was 80. The blood pressure was 140/90.

The basal metabolic rate was minus 10.

Sclerosis of coronary arteries with intermittent angiospasm was diagnosed.

On Aug. 1, 1934, total thyroidectomy was performed. Mr. B. stood the operation well and convalescence was normal. Blood pressure preoperative was 140/90; postoperative, 180/108. He was propped up in bed on the sixth day; he was allowed up in a chair on the ninth. He walked on the tenth day and left the hospital on the eleventh. Six weeks after operation he reported that he felt "like a new man": he had had no attacks since the operation, except that one time after walking too far he had had a slight pain which lasted about three minutes. He was walking outdoors daily, though he had not resumed business. His weight was 193 pounds, his blood pressure 162/120; his pulse was 72.

The basal metabolic rate was minus 26.

On March 29, 1935, eight months after operation, Mr. B. felt much better, he was taking limited exercise daily. His appetite was good and he was sleeping without sedatives. He had had no nitroglycerine, morphine, chloral hydrate or amytal since the operation. He weighed 204½ pounds. The heart rate was 72, the rhythm regular, the sounds were normal. The blood pressure was 172/100. His face and hands were slightly puffed.

The basal metabolic rate was minus 36.

He was advised to walk more, but on level ground and a quarter of a grain of thyroid extract daily was prescribed.

Case 2. Mr. C., aged 53, the manager of two cotton mills, in September, 1934, had sudden and severe substernal pain which was relieved by morphine Since that time he had had repeated attacks, averaging one a week. The pain would begin in the precordial region and extend down the left arm. These attacks usually occurred after a day of hard work or worrying. They would be relieved by morphine. He had not noticed any palpitation nor increase in heart rate. Rest in bed apparently would prevent attacks. He had been nervous and highstrung all his life and had always worked hard, but he had been in fairly good health previously except for "stomach trouble" over twenty years. An electrocardiogram on Sept. 7, 1934, exhibited low voltage in all three leads. A second on Jan. 12, 1935, was essentially similar. Dr. T. L. Ross interpreted them as indicative of "Severe myocardial fibrosis and degeneration." He was admitted to the hospital by ambulance.

Mr. C. weighed 180 pounds. The heart was not enlarged, the rate was 80 and regular, the sounds were of weak muscular quality. The blood pressure was 118/76.

The basal metbolic rate was minus 7.

Angina pectoris was diagnosed.

On Feb. 5, 1935, total thyroidectomy was performed. Convalescence was uneventful, except that the patient was a neurotic and gave interns and nurses considerable trouble. He improved rapidly in spite of himself. He was propped up in bed on the sixth day, he was up in a chair on the tenth day, and he walked on the twelfth. He left the hospital on the thirteenth day after the operation: the basal metabolic rate that day was minus 26.

Seven weeks after the operation Mr. C. reported by letter, "I went to work March 1 instead of April 1, as you advised. I worked four hours daily

at first and then stepped up to six. I haven't had an attack since leaving the hospital, and I sleep nine hours each night without nerve sedatives."

I have been unable to get him back for further observation. He is the type of patient who disregards all advice and probably will be imprudent and have a recurrence of symptoms.

SUMMARY

1. The anginal syndrome is a functional disturbance occurring usually in patients with coronary disease and the "spasmogenic aptitude." Its immediate cause is transitory ischemia of the myocardium.

2. Until recent years angina pectoris was considered a medical problem. Surgery has attempted to relieve the painful attacks by cervical sympathectomy and by alcohol injection of the thoracic sympathetic ganglion. These procedures have proved somewhat dangerous and the results unsatisfactory.

3. Subtotal thyroidectomy gave only temporary relief as the basal metabolic rate was soon restored to normal and the anginal attacks recurred.

4. Total thyroidectomy gives immediate and prolonged relief in the majority of cases, apparently because the lowered metabolism has decreased the demands upon the heart.

5. If the resultant myxedema becomes annoying, small doses of thyroid extract may be given, being careful not to restore the metabolic rate to normal.

6. The technic of total thyroidectomy is more difficult than that of the usual subtotal operation on account of the necessity of coming in direct contact with the recurrent laryngeal nerve and the parathyroid bodies. These important structures are occasionally injured and a surgical tragedy may result.

7. The end results are most encouraging. Taking the cases of Cutler, the cases of Mixter, Blumgart and Berlin and my own, fifty-four cases in all have been reviewed. There were only two deaths in the combined series (less than 4 per cent), a mortality surprisingly low considering the risks involved. The majority of the patients were relieved entirely of attacks, though others experienced greatly modified ones. Many have resumed their business activities under certain restrictions.

8. Total thyroidectomy for the relief of angina pectoris is of too recent an origin to give statistics as to longevity as compared with cases treated medically in the past. The patients generally consider it a success and are satisfied and grateful.

9. While the operation does not remove the underlying cause, it apparently moves the hands of the clock back for the individual patient, giving him additional time and increased activities with relief from pain, provided he cooperates by taking proper care of himself.

10. It is most important to have the closest collaboration between the internist and the surgeon in the management of these patients both before and after operation.

Doctors' Building.

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MANAGEMENT OF THE TOXIC GOITER

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A REVIEW of the history and progress of thyroid surgery during the last 50 years reveals one of the most interesting and gratifying accomplishments of modern medicine. The physiology of this gland has been revealed, classification of the different types of goiter clarified and a technic perfected that makes thyroidectomy relatively a safe procedure. During this process of standardization, many problems and difficulties have been encountered and most of them have been overcome. One of the conspicuous remaining problems is the management of the case of highly toxic goiter or the bad risk.

The purpose of this discussion is to emphasize certain points that are known to all of us doing goiter surgery and to outline a plan of procedure for the management of the highly toxic or bad risk case. I have drawn freely on the writing of Lahey, Frazier, Plummer, Bartlett, Waterworth and others, but the material from which this paper has been prepared is drawn for the most part from my observation, treatment and operations in my own practice during a period of fifteen years and reference is made both to the medical and to the surgical experience during this period.

In summarizing my observations with reference to the preoperative treatment and postoperative care of the highly toxic goiter, I am aware that there is but little that is original or unknown to the majority of you. We learn more about the course of a disease and its management from a review of our experiences than in any other way, especially when that experience has been tinctured with mistakes. This paper is merely an effort to direct your attention to those procedures that in my experience and in the experience of others have proved helpful in managing the bad risk in thyroid surgery.

Viewed in the light of our present knowledge of thyrotoxicosis, and based on the recognition of certain fundamental facts as to the nature and progress of the disease, we are in-

Read before the Sixth Annual Assembly of The Southeastern Surgical Congress, in Jacksonville, March 11, 12 and 13, 1935.

clined to the conclusion that the individual that becomes thyrotoxic presents a characteristically typical background and conforms to a distinctly and easily recognizable type. Just as during the late war we recognized a type of individual with an emotional instability that under strain and stress developed shell shock, I believe that if we analyze the thyrotoxic cases we shall find that they have a characteristic history. They have with few exceptions been nervous children, impulsive, and showing a tendency to emotional instabilities of varying degrees. They have lived intensively and have attacked life at an unrelenting pace without any periods of relaxation. Many years ago Tom Williams called attention to a syndrome found in many individuals and characterized by palpitation, precordial pain, choking sensation and a smothering feeling. Individuals with such a syndrome may or may not develop goiter, but it is very important to differentiate them from the true thyrotoxic individual, as surgery on the thyroid in this type of individual is fraught with pitfalls; and, even if goiter develops, removal of the gland may not be effective in restoring the patient to normal or to alter his vagotonic characteristics. The true thyrotoxic patient is prone to typical remissions and exacerbations, but it is usually in the relapsing phase that he presents himself for surgery. It would greatly lessen our anxiety and responsibility if these patients would apply for surgery during a remission, but unfortunately it is only occasionally that one cooperates to this extent.

In my experience over a period of fifteen years I have operated on patients of practically all ages from 10 to 69. The majority has been between 25 and 45 years of age, but the overwhelmingly toxic cases have been between 30 and 45. During this period there have been nine deaths following operation, and all, with one exception, occurred in thyroid crisis. This case I attributed to reaction to procaine as the patient died on the table immediately after the skin had been infiltrated with local anesthetic. All the deaths were in cases of toxic adenomas. There has been one severe postoperative hemorrhage which fortunately was discovered and controlled before it assumed alarming proportion. There have been no permanent nerve injuries. There developed two cases of postoperative tetany, which were satisfactorily controlled and responded promptly to calcium and Collip's serum.

If we are to plan our management of the thyrotoxic case effectively and intelligently, it is necessary to preface the discussion of the treatment of these cases, namely, the plus four toxic goiter, by stating what is meant by the term and to classify these cases accordingly. By "plus four toxic," I refer first to the acutely toxic, diffuse goiter which may or may not be associated with exophthalmos and is most frequently encountered in patients younger than those with the toxic nodular goiter. In my experience and in the experience of others, this condition has been found most frequently in patients from 12 years of age up to the early forties—the incidence being greater in the early thirties. Second, there is the acutely toxic nodular goiter. This occurs in patients who, as a rule, are older than those with exophthalmic goiter. It is in this type of case that a nodular goiter present for many years, probably had not given any serious trouble until suddenly there is an onset of toxic symptoms. This type is usually encountered in patients from 35 to 50 years of age. In the third type acute symptoms of toxic goiter develop suddenly in pregnancy, or with an infectious process, perhaps of the tonsils or sinuses. There is a fourth type which has been referred to by Lahey as atypical goiter in which the symptoms appear to be neurocardiac. Crile has emphasized that often thyroidectomy produces no improvement in this type of goiter, and maintains that adrenal denervation does much to stabilize and rehabilitate these individuals.

It is in the preparation and management of these different types of cases that frequently the ingenuity and judgment of the surgeon is challenged. In the average case of toxic adenoma or exophthalmic goiter with a moderately increased metabolic rate, that is, constantly below plus 35, I feel that the patient will respond to the ordinary routine treatment of rest, preliminary sedation and the judicious administration of some form of iodine. It is not to these milder toxic cases that I invite your attention, but to the large class of more severely toxic character that is regarded as one of the unfortunate medical and surgical problems, and in which intensive treatment and wise surgical judgment are demanded.

The preoperative use of iodine has reduced considerably the mortality of Graves' disease, and it was earlier thought by some that iodine would control even the most toxic cases.

However, this idea is not entirely correct. Lahey has stated that in a series of 1,118 patients treated for goiter in a year's time the mortality rate from thyroid crisis equaled the post-operative death rate for patients of all types of goiter. If this is accepted as a clinical experience, the conclusion is that we must not place our entire reliance on the use of iodine as a pre-operative measure. Not only must proper measures be instituted before the operation, but the proper time selected and the proper type of operation performed if we are to insure our patient his best chance of recovery.

If this patient is regarded as a bad risk, I prefer to have him in the hospital for a few days prior to operation, the length of time depending on the severity of the symptoms. He is kept as quiet as possible, sedation is resorted to if necessary, and the basal rate is then again determined. If the rate remains high and shows no tendency to regression, I again insist on a few more days' treatment and observation. If there is no improvement and it is economically undesirable for him to remain in the hospital, he is allowed to return home for intensive iodine therapy and rest in bed for 14 to 18 hours a day for two weeks. It is my opinion that more than two weeks preliminary operative treatment gains nothing, for these patients have reached about the maximum improvement in this length of time.

After this preliminary treatment, the patient is again subjected to a careful check-up and decision is made for or against operation. If there has been no improvement in metabolic rate, no increase in appetite or gain in weight, no tendency towards stabilization of the nervous system, or if fibrillation is present, further treatment and observation are required. In the toxic case probably the most important consideration is a gain in weight. Of course, the metabolic rate is an important consideration in intelligently determining the management of a given case, and I should be reluctant to operate in the presence of a rising metabolic rate, even if the patient evidenced an increase in weight and an improvement in general nervous stability. Fibrillation does not denote an absolute contraindication to operation in a patient otherwise stabilized, but one can operate with a greater sense of security and a feeling of lessened hazard if this symptom has abated.

These highly toxic patients should have a complete medical

survey, and the closest cooperation with the internist is necessary. A critical analysis of kidney function, heart action and blood chemistry is valuable. If these cases presenting advanced symptoms are carefully analyzed, it will be found that a certain number of the patients will die while under observation and that over a period of years this percentage will compare with those dying immediately or a few days following operation from a postoperative thyrotoxic crisis.

I have found the intravenous administration of glucose for short periods of time preoperatively of great value. Frazier's investigations on carbohydrate metabolism in hyperthyroidism have revolutionized the management of these severe cases, and they are the most important contribution therapeutically since the epochal establishment of the administration of iodine on a sound, scientific basis. The amount of glucose solution or other fluids administered intravenously must be determined by the record of intake and output, and be stopped on the appearance of edema. A well-balanced diet is indicated, and I have personally found the administration of honey along with the diet of distinct value.

We have less to fear in the exophthalmic than we do in the toxic adenomatous type. We have in Lugol's solution or some form of iodine a drug that is of positive value in the preparation of the exophthalmic case; whereas, in the adenomatous toxic type, the use of iodine is not of striking value preoperatively.

In estimating the operative risk probably the most important criterion is the weight curve; for instance, an individual that has lost 20 or 30 pounds during the previous year, but has maintained his weight for the previous six months is not a particularly bad risk. On the other hand if during the preceding month or so the patient has lost 15 or 20 pounds, he should certainly not have an immediate operation. If operation is done, it should be confined to single lobectomy. Another important criterion is muscular strength, and in estimating the patient's ability to stand operation this must be considered. Creed has stated that a woman who has sufficient strength to do her own housework has sufficient strength to go through a thyroid operation. However, with the more serious cases other methods of evaluation must be resorted to. If the patient has been sufficiently iodinized, there are certain

cases, especially of the exophthalmic type, in which one might operate on estimation of the factors of weight and strength alone.

While the basal metabolic rate is one of our most valuable aids, it may be of little value in estimating the operative risk in any given case. As a rule basal metabolic rates above plus 75 increase the risk and are not therefore favorable; on the other hand, those much below that rate may or may not be good. It is the proper interpretation of the reading, the correct estimation of the reaction of a given individual, and not the height of the metabolism that is important.

One of the most important observations to my mind both from the standpoint of treatment and also of determining a patient's ability to stand operation, is nervousness or restlessness. It is not always true that the severity of the tremor is proportional to the degree of nervous stability, but it is my feeling that you can almost with certainty foretell the reaction of certain patients from the degree of their nervous instability. One of the most important contraindications to operation is restlessness, and earlier in my experience failure on several occasions to interpret this one observation correctly led me into pitfalls that were later viewed with regret.

In most cases the pulse rate is not of much assistance, as in many of these very toxic cases the pulse rate often varies without much regard to the intensity of the illness. However, a persistently high rate is not indicative of a safe risk. All of us doing much thyroid surgery realize that the worst time to consider operation is when the patient is on the decline, or approaching a crisis, as indicated by nervousness, increase in pulse rate, sleeplessness, and loss of appetite. Anorexia is a significant warning, and I am convinced that we are inviting disaster when we operate on a patient who does not have a good appetite. It is not only bad surgery to operate on a patient approaching a crisis as indicated by symptoms, but it is equally bad to operate too soon after the crisis. A period of from 6 to 10 weeks should elapse after a crisis before surgery is attempted, for it is desirable to allow time for the development of another crisis if it is imminent.

There are many factors involved in determining the best time to operate. But there is the advantage that when we have evaluated the case and realize that an operation is attended

with dangerous possibilities, we can plot our management accordingly and confine surgical interference to ligation or the removal of one lobe at a time. There will be occasional patients who can never meet the requirements for operation. They are apparently destined to die and will only succumb earlier if operated on, and I am in agreement with the dictum laid down by Bartlett that any patient dying within 72 hours after operation indicates that we have failed in our evaluation of the patient's ability to stand whatever was planned, or too much surgery has been done at the wrong time. In determining when to operate, I consider the following things as indicative of a favorable prognosis: if the patient has been intelligently iodinized, or if there has been a gain of a few pounds or at least no recent loss of weight, if his muscular strength conforms to the previously mentioned requirements, if his metabolic rate has been properly estimated and is not rising, if he has no edema and can walk a reasonable distance without becoming short of breath, and if he is able to hold his breath for more than 15 seconds. With these things in favor of the patient, I feel that operation can be undertaken with a reasonable degree of safety.

In considering the use and abuse of iodine in the treatment of goiter, we are confronted with much conflicting opinion. Its usefulness in the preparation for operation has been established beyond question, but its widespread and indiscriminate use has been a menace. Iodine is not a cure for toxic goiter and should be used only as a measure preparatory to operation. There are many cases in which its use fails to induce the characteristic remission of symptoms. In some cases it appears to have no effect upon the disease. Its administration may even accentuate all the symptoms which are present. A decision when and when not to use iodine will be based largely on clinical experience, upon observation in the individual case, and upon consideration of all the symptoms and findings presented. In thyroid surgery the personal equation, the individual factor judged by observation and experience, is often the factor determining recovery or death.

There is a certain percentage of cases in which we do not find any reaction to iodine, and this class of case has been referred to as the iodine resistant thyroid. It is in this type of case that the fractional or stage operation produces the safest

method of approach. The greatest concern is caused and difficulty encountered in those cases where the metabolic rates remain persistently at 50 or above. About 10 per cent of the exophthalmic group will fail to benefit, or may even be made worse by the administration of the usual dose of 10 or 15 drops of iodine three times daily, over a period of from ten to fifteen days. Long-standing symptoms and a history of prolonged use of iodine often explain this tendency to the refractory state. Fractional operations should be more often done in this type of iodine resistant hyperthyroidism. This is specially indicated if the patient is an elderly or fragile individual with marked cardiac damage and long-standing thyrotoxicosis. A paradox repeatedly emphasized is that iodine, so important in reducing the mortality of thyroidectomy, may by its injudicious and indiscriminate use be the factor which makes operation unusually hazardous, and as a result surgery can only be feasible or resorted to safely when done in stages.

In the adenomatous type of goiter it has been my experience that the use of iodine in the toxic group does not produce the striking improvement that it does in the exophthalmic. In fact there are many cases in which there is no reaction whatsoever, due in most instances to the replacement of the active acini by areas of degeneration incapable of utilizing the iodine administered. However, this fact has been observed, that by the use of iodine in adenomatous cases, we can predict with some degree of accuracy what the postoperative reaction will be. If before operation we observe a marked response to iodine, we can have some assurance that the postoperative course will be happier and there will be less likelihood of severe storm. I have for this reason routinely used iodine in preparing cases of toxic adenoma for operation. The incidence of iodine resistance in the adenomatous group is probably twice as great as that noted in the exophthalmic group; consequently, I feel that the fractional or multiple stage operation is more often indicated in this type of goiter than in the exophthalmic. These observations lead me to the conclusion that iodine must be used judiciously in toxic cases and always with the view to operation at an early date.

Operation cannot be considered in this paper except to insist that the type and extent of each operation should be carefully planned beforehand, and that procedure should be used

which, in the judgment of the operator, best suits the requirements of the individual case. It is important that too much surgery should not be done at one time, and when we are dealing with doubtful operative risks, experience dictates that it is best to divide the operation into two or more stages. This has been emphasized by Clute, who states that "The degree of postoperative reaction in severe hyperthyroidism is exactly related to the amount of surgery performed in each case." Local anesthesia has been employed in 90 per cent of my cases. Nitrous oxide and oxygen is my preference when general anesthesia is required.

The postoperative care is of utmost importance as it is difficult to predict just when a disastrous storm will be encountered, and intensive treatment should be begun early. For this reason anticipation is better than regret, as there are no positive criteria for selecting the cases that will have a stormy postoperative course.

The most useful measures for the control of postoperative crisis are iodine, fluids and drugs which control restlessness and delirium.

Morphine and sodium amytal have been most frequently employed for the control of restlessness and delirium. These patients tolerate large doses of morphine, and I have not hesitated to give it freely, supplementing it when necessary with sodium amytal or nembutal by mouth or rectum. I have on occasions used one or both of these drugs to the point of narcosis.

Fluids are essential, and it is amazing the quantities these patients require and can handle without edema. Saline is given routinely by hypodermoclysis, and I start these patients immediately after operation on continuous venoclysis of glucose in saline. A recent patient was given 5,600 c.c. by venous drip every 24 hours for three days. This is probably our most useful measure in combating high temperatures and controlling the rapid pulse rate. Large amounts of fluid by mouth and rectum can also be given. In 1933 Frazier reported that since the adoption of the glucose venoclysis method of management that there had not been a single operative death on his thyroid service in a consecutive series of 535 operations for thyroid disease, and that the number of days elapsing from

the time of operation to the return of a practically normal afebrile state had been reduced materially.

Iodine used postoperatively will often prove an added weapon in combating or preventing a fatal crisis, and it may be safely used in both the exophthalmic and the toxic adenoma group after operation. It is routinely given by mouth or by rectal drip. We have not used any form of iodine by hypodermoclysis, but many writers state that it is well tolerated in this manner. In very severe cases I do not hesitate to give Lugol's solution intravenously in saline, and have noted uniformly good results. From 20 to 50 minims in 300 c.c. of saline may be used freely with impunity. In a recent case I gave 160 minims during a 24-hour period intravenously with a very striking response. Goodrich has reported splendid results from the intravenous use of sodium iodide. Experience during the past few years has indicated that Lugol's solution can be effectively and safely given intravenously, and has unquestionably been a useful and additional measure in handling thyroid crisis.

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The Southern Surgeon

Published Bi-monthly by

The SOUTHERN SURGEON PUBLISHING COMPANY

478 Peachtree Street, N. E.

ATLANTA

Subscription in the United States, $3.00

VOLUME IV AUGUST, 1935 NUMBER 4

THE CLINIC AND THE COUNTRY DOCTOR

WITH the turn of the century the hybrid medical school dominated by commercial interests felt the heavy hand of the Council on Medical Education and Hospitals of the American Medical Association. This survey focused attention for the first time in this country upon the fact that medical schools justify their existence largely through their devotion to the public interest. Then was inaugurated the policy of careful selection of applicants for admission to their classes—students actuated by high purposes and qualified by a background of cultural and academic education. With this emancipation of medical education and medicine from its trend toward a business came a new appreciation of the function of school and product—a rededication of both to the advance of the science and art of medicine to the end that human disease might be prevented, its ravages ameliorated, or effectively treated. It followed as a natural sequence of such catholicity of medical thinking and motive that not only was better training in medical schools necessary but the graduate, too, must be reached, and the public made health-conscious. To supplement the requirements of post-graduate instruction, medical societies, na-

tional, regional and state, have committed themselves, in principle at least, to the task of keeping their membership familiar with the advances constantly being made in theory and practice; and public health agencies have multiplied to keep pace with the interested demand upon the part of those for whom in its final analysis the benefits of medicine are conceived.

As past progress has followed a wider diffusion of knowledge, so will the twilight zones of medical practice be illumined when the clinic is carried to the rural practitioner. While one would be bold to assert that the physician of the populous community was perhaps oversteeped in the cauldron of frequent medical meetings, it can not be gainsaid that the family doctor who sees first most of our physically handicapped, has not enjoyed a commensurate degree of opportunity for practical instruction. If, as many believe, there is over-organization in the profession and overlapping of effort in post-graduate instruction, this criticism can only be maintained with respect to the urban physician along with that remnant from town and country who frequent our oft-recurring assemblages.

The opportunity for quick results lies not in more elaborate city meetings but in decentralization of effort by carrying the clinic to the country doctor who is ready and eager to listen to practical instruction and anxious to supplant his obsolete methods by new and more effective procedures.

While there may be ready agreement on this necessity among those who shape the policies of the extension departments of medical schools and formulate the activities of medical societies, the fact remains that no sustained program in this direction has been prosecuted in the territory covered by our Congress. Indeed, one is wont to inquire if it would not be to the interest of the profession throughout the country if some official body carrying the same weight of authority as the Council on Medical Education and Hospitals was created to make a survey of medical organizations, looking to the elimination of those in a given territory whose activities are limited to the holding of annual meetings in which quasi-scientific papers are presented. Something more is necessary if a medical society is to earn public approval and make claim to the active support of the profession. It is a question of quality of organization rather than number, of mission rather than name,

of objective rather than location, of fruit rather than embellishments in personnel and program.

It was with such fundamental consideration in mind that The Southeastern Surgical Congress was founded. From its beginning it was dedicated to teaching. For its faculty it seeks to include all those physicians who measure up to the four-square standard—unquestioned character, sound training in the art and science of practice, an appreciation of the dignity and obligation conferred by membership in a learned profession and a willingness to contribute through the written page, the platform, or the clinic to the higher development of Southern doctors and the elevation of the standards of Southern practice. Their classrooms, we believe, should be set up in town and hamlet; their material the patient presenting practical problems of interest to the family physician. Our teaching will thus attack the problem of post-graduate education in the neglected community where labor the great army of the unobserved to whom all medical learning in its finality must go for effective application to the patient.

To this end the Executive Council of the Congress has approved and recommends the holding under state chapter auspices of frequent, one-day clinical programs in rural districts. The clinicians, for obvious reasons, should, whenever possible, be drawn from the immediate territory concerned with the discussion supplemented by visiting colleagues. The patients presented will be selected so as to furnish for analysis the commoner problems met by the rural physician.

To such meetings where post-graduate instruction shorn of its pomp is presented in simple fashion, there will come an earnest group moved little by the erudite methods which compliment the metropolitan amphitheatre, but quickly responsive to the teacher who has a practical message.

In such soil we will do well to plant. From such planting in due season the abundant harvest will be reaped. With such a program The Southeastern Surgical Congress will deserve to be included in that family of societies which by the very excellence of their services to the public have earned for themselves the esteem and gratitude of the community.

C. W. ROBERTS, M. D.

BOOK REVIEWS

The Editors of THE SOUTHERN SURGEON *will at all times welcome new books in the field of surgery and will acknowledge their receipt in these pages. The Editors do not, however, agree to review all books that have been submitted without solicitation.*

DISEASES OF THE THYROID GLAND. By ARTHUR E. HERTZLER, M. D., Chief Surgeon, Halstead Hospital; Professor of Surgery, University of Kansas. Pp. 348, with 181 illustrations. Third edition, entirely rewritten. Price, $5.00. St. Louis: The C. V. Mosby Company, 1935.

The SURGEON has maintained more than once that a surgeon who practices in a relatively small community with a stable population and thus has an opportunity of following his patients over a long period of years and of re-examining them as he desires, has an unrivaled opportunity to advance medical science. Such a surgeon is in the ideal position to do scientific clinical research, perhaps after all the most important type of investigation. Hertzler is the exemplar par excellence of this school.

His new book on the thyroid, rewritten from beginning to end except for two paragraphs, Dr. Hertzler emphasizes, "is an expression of my own experiences and impressions in dealing with affections of the thyroid gland." His most startling conclusions are:

1. "The continued observation of many of my old patients has brought out more forcibly than ever the fact that the disease of goiter is a continuous process the normal termination of which is a cardiac death."

2. "Experience has tended to more and more radical operation culminating in complete thyroidectomies in certain selected cases. Myxedema has not resulted."

3. "The basal metabolic reading . . . is only one factor and a minor one, when one considers the thyroid disease as a whole. The acceptance of this test as a measure of all the mischief the disease may do the patient is responsible for most of our misconceptions regarding the nature of goiter."

There was a time when the reviewer would have considered a man holding such views, to use one of Dr. Hertzler's words, "haywire." Careful reading of this book, though he may not have been completely converted, has convinced him that Dr. Hertzler is approaching the truth.

Since a work on pathology of the thyroid is in preparation, this phase of the subject does not receive as much space as in former editions. The book otherwise leaves little to be desired either in illustrations or information. The author has learned the presentation of scientific data is not injured by the exhibition of common sense and wit. Indeed he and Will Rogers are this reviewer's favorite living authors. But why, oh, why did Dr. Hertzler three times avail himself of that barbarism "diagnosticate"?

THE CLINICAL MANAGEMENT OF SYPHILIS. By ALVIN RUSSELL HARNES, M. D. Price, $1.50. New York: The Macmillan Company, 1935.

The physician who is not particularly interested in syphilis will find in this book a definite authoritative outline of modern treatment. For those who like outlines, the book will prove satisfactory.